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TO THE MEMORY

OF

HARRIS P. MOSHER

1867-1954

TEACHER, AUTHOR

INVESTIGATOR AND

LEADER

THIS ISSUE OF THE

ANNALS

IS REVERENTLY

DEDICATED BY HIS

FRIENDS, FORMER STUDENTS

AND COLLEAGUES

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I

HARRIS PEYTON MOSHER, M.D., D.Sc., LL.D.

FREDERICK T. HILL, M.D.

WATERVILLE, MAINE

Recipient of every honor possible and recognized throughout the world as one of the outstanding leaders of all times in otolaryngology, Dr. Mosher's accomplishments in the field of his chosen specialty are so well known that any attempt at enumerating them here would be unnecessary. His contributions to the literature and to education, together with the result of his researches will ever remain a monument to his memory.

But what of the man himself? For this Memorial number of the Annals I should like to portray the personal side of his life, the underlying characteristics that made him what he was. In trying to paint a word picture of Dr. Mosher I may violate one of his dictums, never to apologize for what one is about to say; that if apology is warranted one had best keep silent. However, what follows may seem to some as having too much of the personal, too much use of the first person. To this I can only say that to describe the real Dr. Mosher as I know him, requires the employment of the personal approach. For this, I beg the reader's indulgence.

One of our popular periodicals for years has featured an article entitled, "The Most Unforgettable Character I've Met." This could be applied most aptly to Dr. Mosher. For, indeed, he was "most unforgettable" to any otolaryngologist who ever had known or worked under him. His entire life history, his motivation, his underlying

philosophy makes an intriguing story, a story of a most human, sincere person, activated by the highest of ideals. To those who knew him less well and, thus perhaps failed to understand him, he often seemed an enigma.

It was the good fortune of the writer to have known Dr. Mosher rather intimately for some 40 years, first as a student and house officer, then as a disciple, and finally as a close friend. Ever since his retirement, up to the time of his death, we carried on an active correspondence on the average of once a week. We travelled together to medical and Board meetings. We visited in each other's homes. And we exchanged confidences, some too personal to divulge. Going through his letters, which I had saved, after his death, I found in one the request that when the time came, I write his obituary and, characteristically, the phrase, "for Heaven's sake put some life in it." I have tried to follow this admonition as best I could, for the Annals and our two senior societies, but it proved to be one of the saddest of duties, for it marked the close of a treasured friendship.

My first personal encounter with Dr. Mosher had been far from auspicious. At that time I was the "low man on the totem pole" of the house staff at the Infirmary and as such the laboratory work was a part of my duties. Dr. Mosher had brought some specimens from a private case which I was only too glad to run through for him. The trouble came when he handed me money in payment and I tried to refuse. Then did I get "my ears burned off" and learned in no uncertain terms who was boss. This is mentioned only as an example of what often was the experience of younger men in their first contact with Dr. Mosher.

At that time he was far from the dominant figure he later became, being simply the head of one of the otological services. But, even to a callow and inexperienced house officer, his service had an intangible something. It was hard and militant, but nobody worked harder than the chief. He was indefatigable in everything he did. He expected and got the best out of his men. There was a spirit of inquiry, of research, even with apparently simple clinical problems. Ever he was the teacher. As a result, house officers looked forward to his service, realizing what would be expected of them and prepared for criticism whenever warranted. He had much to give and was generous with his giving. He always wanted everyone to be able to do everything he could do and never tried to hold anyone back. But he demanded sincerity, loyalty and industry.

He himself had come into the specialty, "through the back door." He had not had a residency in otolaryngology. His hospital training

had consisted of a surgical interneship at the Massachusetts General Hospital and one in obstetrics at the Boston Lying-In. After serving as a contract surgeon in the Spanish American War he had spent four years in general practice in Boston. During this period he got his first taste of otolaryngology in the out-patient department of the Massachusetts General Hospital. Deciding upon the specialty as his future career, he had taken postgraduate work in Berlin and Halle. Returning home he resumed his connection with the laryngological service at the General and became associated with the Massachusetts Eye and Ear Infirmary. Here he literally fought his way to the top, not in a combative sense but by determination and hard work. From his beginning in practice he had taught at Harvard Medical School, first as an assistant in anatomy. Boston medicine and the Infirmary in those days were rather provincial and conservative. He was not a native Bostonian, although he had acquired a home on Bacon Street, as he said, "at the wrong end." The path upward was not an easy one but that never deterred him. Many years later, at a meeting of the American Board there was a discussion as to the necessary qualifications for candidates. Dr. Mosher who was presiding, asked how many of us were self-trained. When no hands were raised he said, "Well I guess I'm the only one." This indicated his own frankness and, most important, the fact that one's education in medicine is never completed and that self-education, if continued, could be of greater value than too much reliance upon what he termed "breast feeding."

In the early days at the Infirmary he had the support of one of the older surgeons, Dr. Plummer. He was a most interesting person. A rugged athlete and life-long friend of John L. Sullivan, he also was an outstanding Greek scholar. Toward him Dr. Mosher always exhibited that strong loyalty which was so characteristic. In his remarks at the 125th Anniversary of the Infirmary he said Dr. Plummer had given him his first chance in the institution and closed with the following paragraph:

"Bv some members of the Staff, there was a raising of eyebrows at Dr. Plummer's striking personality. He had the courage to be himself at all times, a thing which many of us would not dare to do. Those who lifted their eyebrows, I rated then, and still do, far below him as a man."

Dr. Mosher gave the same unswerving loyalty to two other older Boston otolaryngologists, Dr. Algernon Coolidge and Dr. Frederic Cobb whom he termed his godfathers in the Specialty. This characteristic was not limited to those who had taught and helped him in his younger days, but was given freely to every confrere of his own

or younger age group who met his personal standards. And once given, it never was taken away, as many can testify. Dr. Mosher set his standards high, both for himself and his friends. Sometimes this was not understood and gave rise to false impressions.

Many feared his criticism. But when he felt criticism was justified, it was forthcoming—no matter when, or where. He was completely honest and frank, and this, combined with an intense abhorrence of hypocrisy, impelled him to denounce, in no uncertain terms, anything that smacked of dishonesty or insincerity. He was dedicated to medicine and felt a compunction to destroy any assumption that was false or misleading. It mattered not if these be offered by his own friends or associates. At the same time he would as quickly defend his own worst enemy when he felt he was right.

I recall one day after the close of a meeting in Atlantic City. Dr. Mosher had waxed most sarcastic in the discussion of a paper by a man he personally admired. Actually it was a matter of priority of a technique and the claim of the reader clearly was incorrect. Walking back to the hotel Dr. Mosher said he wished he had not been so violent in his denunciation. He said it was due to diffidence and that he simply could not avoid becoming sarcastic when he "hated to have to do something."

Another time a younger man, who had never hesitated in showing his animosity towards Dr. Mosher, had been proposed for membership in one of our senior societies. Dr. Mosher had been out-spoken in behalf of the candidate. Asked by a mutual friend why he had endorsed him, Dr. Mosher replied that although he did not like him personally, he felt he was a worthy candidate and should be elected. Along this same line I know of at least two men whom he kept out of a senior society, not because of poor professional qualifications, but because of their habit of telling what he called "smutty stories" in public.

Does this seem paradoxical? It would not to Dr. Mosher. To achieve the heights in medicine, to him, demanded not only professional skill, honesty and good character, but the attributes of a gentleman. This did not mean money or social position, but just certain niceties of everyday conduct.

To really understand Dr. Mosher one must go back to his heritage and his early life. Born in Woodfords, Maine, now a part of Portland, he came of old New England stock, being a descendant of Paul Revere's sister. His father, Andrew Jackson Mosher, was a civil engineer and something of an inventor, being largely responsible for the development of the United Shoe Machinery Corporation. From

him, the future physician inherited a mechanical ability reflected in the numerous surgical instruments he later developed, as well as financial resources sufficient for him to devote his talents to research and teaching without the necessity of carrying on a private practice. His mother was Julia Harris Woodford Mosher. A talented musician, she must have been a devoted mother and a very wonderful woman, for her influence was a paramount factor throughout her son's life. He once told me his earliest remembrance was of being carried in his mother's arms to the choir loft of the church where she was the organist. In later life whenever distraught or worried it was his habit to repair to the third floor of his home where he kept her Estey organ. There he would play some of the old hymns that were her favorites. This always was a solace to him. Her influence must have been responsible for many of his sterling qualities, the obligations of good citizenship, patriotism, and gentlemanly behavior. Perhaps what some people considered a somewhat puritanical attitude towards certain types of so-called humor, may be attributed to his early upbringing.

I am sure that this accounted for a certain reserve he maintained throughout his life towards his confreres and friends. I know of only two persons who ever addressed him as "Harris," his wife and one medical colleague, and I feel that in this latter case, this really annoyed him. Yet he actually missed the warmth and congeniality that somehow is associated with the use of the first name among friends.

The reason for this became clear to me in the later years of his life. As before mentioned we had been corresponding regularly and always with the formal salutation of Doctor. One day he wrote me to the effect that our friendship was a close one and suggesting that we address each other more informally. I was away at the time and the letter was delayed in reaching me. Before I could answer I got a second letter asking me to please take this seriously. He said that first names did not come easily to him and added something which told me a lot. He said never in his life had he heard his mother address his father as other than "Mr. Mosher." When I wrote back that I'd always thought of him as "The Chief" and, with his permission, would address him as such, he answered that it pleased him to be still thought of as the chief.

Incidentally although he rated many titles, ranging from Colonel to Professor, the one he liked best was "Chief." This seemed to represent those things in his professional life that he treasured most, his teaching at the school and his service at the Infirmary.

To many who did not know him or who had only casual acquaintance he often seemed gruff and even disagreeable. Certainly he never was loath to put one in his place, if he seemed to warrant it. And looking back on many such an occasion when this happened to me, I know that it was called for and was for my own good. He suffered under a sort of complex that it was his duty to correct any deviations from what he considered as correct and proper. Again the old New England concept of familial discipline. For in many ways, otolaryngology was his family.

Yet, underneath it all, he was the most kindly, generous, human of persons, with more than a little sentiment, which he always tried to conceal. Only in rare moments of personal confidence, or in his letters, would this come out. No one will ever know the number of young men he helped to get a medical education or training in the specialty, of the many people in need who were objects of his charity. At the same time he had little use for those who paraded their charitable acts. One remark of his seems worthy of quotation here: "Philanthropy is all right as an avocation but bad as a vocation."

One woman, other than his mother, and only one, likewise played a most important part in his life. He did not marry young. In fact he was 46 when Mrs. Mosher and he exchanged their marriage vows in the old North Church in Boston. They were the first couple married there after the church had been restored. Their life together could only be described as idyllic. She brought much into what undoubtedly had been a somewhat lonely existence. I have a copy of the dairy he kept during his tour of duty in Europe in the First World War. It is filled with references to his beloved Helen. In fact it was written for her, to supplement letters which might go astray. On the occasion of one of the Board examinations in San Francisco, he and I shared a drawing room on the trip out and back. At every station stop when there was time he sent her a telegram. And every night it was his custom to talk to her by telephone. I recall his saying, "I suppose you think I'm a silly old fool but we are all each other has." Never have I seen greater devotion.

Dr. Mosher had almost a fear of showing emotion, perhaps another manifestation of diffidence. He tried to balance what he called "a sensitive heart with an intelligent mind." When we laid the corner-stone of our new hospital he drove down from Marblehead, a matter of some 400 miles, both ways. Naturally I was touched by this and tried to tell him so. He shrugged off my attempt with a gruff, "You know why I came."

As one of Maine's most distinguished sons in medicine Dr. Mosher was invited as an honor guest to participate in the 100th Anniversary

of the Maine Medical Association, two years ago. While I know it was a chore for him he gave it that same meticulous preparation that he did with all his papers. He even insisted on trying out the public address system before the meeting at which he was to speak. It was my privilege to look after him during his visit and the following day we drove up to my home for him to see the new hospital. In a letter to me on his return home he said that he never before came so near "blowing off my top emotionally as I did while under your care. It hurts my pride to acknowledge it."

Dr. Mosher was widely known for his keenness of mind, his incisive thinking, and his ability to get at the gist of any discussion. He possessed a rare sense of dramatic values which he used to great advantage both in his teaching and in his presentations before medical meetings. Not only were his papers of sound scientific value but they were written in perfect English. And they had been written and rewritten many times before presentation. He used what he called the "scissors and paste" technique, rearranging his material until he was satisfied with the result. And he would be of greatest assistance to others in criticizing their material, as I know from personal experience. How often has he asked, "just what are you trying to say" after listening to some feeble efforts to express a thought. And, when the question was answered his reply would be, "Then, damn it, say it" and one would realize the virtue of directness and the folly of verbosity. For many years we exchanged the first draft of papers for criticism, and while I was always his debtor in this custom, I will say that he expected and demanded frank criticism himself and could always take it.

He recognized the limitations of specialization and always was endeavoring to broaden his horizon, not only in medicine, but beyond. He practiced, and advised for others, "regularly reading something worth-while in non-medical literature." His early training in art not only provided him with a hobby which he followed all his life, but enabled him to develop methods of teaching which were unique. In these his students made drawings and casts from specimens and dissections. He worked in all media. While travelling, it was his habit to make drawings of the landscape from the car window, from which he made water-colors. Marblehead provided him with picturesque material for both sketches and etchings. He was greatly interested in the life-story of Albert Schweitzer and intrigued with his leonine appearance. One of his last pieces of work was a medallion of Schweitzer of which he was quite proud.

He was a delightful companion and loved the fellowship of his friends. Nothing suited him better at the close of a meeting of the

Board, or one of the societies than to sit with a congenial group, and over a scotch-and-soda, participate in informal discussions. These might be in a serious vein but often were light and gay.

He had a strong sense of humor and loved a good joke—if it was a clean one. In his earlier days he often was responsible for various gags, stunts or skits at the banquets of the national societies. One of his famous productions involved the use of lantern slides with the heads of certain dignified members superimposed upon torsos in ridiculous apparel and situations. He was a master of repartee and loved what he termed a "wise-crack." Sometimes when someone said something particularly funny he would remark he wished he had said that. Regarding humor he once wrote that he liked it in a letter and even in a medical paper—"But in a medical paper it must be handled with care, like a new baby."

He delighted in coining epigrams, such as:

"Present day life consists chiefly in finding a parking space."

"In your job, as with your vest buttons, begin at the bottom, or you are liable to miss the top."

Once he sent me a paper entitled: "Thoughts, often cynical, while shaving." Among these the following are revealing.

"Missed opportunities for good or evil last long in memory, and often cause equal regret."

"The front door of every home opens on heaven or hell."

"Men are divided into two classes, those who grasp for all the credit they can reach, but never give any. The other and smaller class, give credit where they see it is due."

" 'All men are born free and equal' was put into the Constitution to catch votes, and has been used for this purpose ever since."

"Go to the funerals of other people if you expect anyone to come to yours."

In a letter received shortly before his death he spoke about his coming operation, about the ravages of the hurricanes and the damage done to his Marblehead home and that he was glad he had been missed at the Board and Academy meetings. He said he was a bit gloomy at the changes made inevitable by Time. And then added the following lines which had occurred to him that morning. "The hunger to be remembered ends only with Life's last breath; and a cold death notice of a line or two marks the hunger's end."

And so died one of God's noblemen, a great physician, scientist, and teacher, certainly one "unforgettable character" in otolaryngology, but leaving to us a great heritage, the results of a life well and truly lived for the benefit of mankind.

Opposite page: with F. T. Hill, relaxing after an excmination in Philadelphia, 1946.

II

HARRIS PEYTON MOSHER AS A TEACHER

LeRoy A. Schall, M.D.

Boston, Mass.

One week before his death, Dr. Mosher restated that he considered teaching his greatest contribution to otolaryngology. Those who knew him well will remember that his one and greatest joy was his contact with his students. This contact did not end in the classroom, but it was the beginning of a friendship which was projected throughout the years.

From the beginning of his career, Dr. Mosher was interested in anatomy. His first appointment at the Harvard Medical School was in the Department of Anatomy, and he held this appointment from 1899 to 1909.

Upon his return from Europe, after having studied with Janssen in Berlin and in Schwartze's old clinic in Halle under Grunert, Dr. Mosher brought back many of Janssen's ideas on sinus anatomy; and he instituted a course on the accessory sinuses. This was the beginning of his well-known graduate course which has continued throughout the years with the exception of interruptions during World Wars I and II. His teaching skill made it one of the best special courses of this type to be given in America.

The course was a concentrated one, and students under Dr. Mosher worked from early morning until late at night, Sundays and holidays included. His teaching was by visualization, by dissection, by tracings and drawings, and by reproduction of specimens in plaster.

In his *Memories of Postgraduate Teaching,* he writes, "My courses featured applied anatomy. I held that it was only if you knew your anatomy were you ready for surgery. The parts of my postgraduate course which I hoped to be of the most lasting value were:

"First, a comprehensive series of drawings of picked anatomical and surgical specimens. A drawing was always started in the tracing box, and then finished accordingly to the student's native skill in drawing and with what help I could give him as he went along.

"Second, a series of casts to supplement the drawings made from plaster casts or from the cadaver. The casts, unlike the drawings,

were in three dimensions and constituted an anatomical and surgical library which ranked next to the cadaver for study and was always available."

Dr. Mosher was happiest when his course was in progress. The lectures which he gave were written and rewritten before he presented them. He was continually drawing, painting, etching, or modeling. His anatomical charts which he made for the course are still in use, and they have dramatically simplified the teaching of sinus anatomy.

From the students he demanded not only accurate dissection, measurements and drawings, but application of the knowledge obtained by their means to the surgical problem of the part being studied. Comparative anatomy played an important role in the course. Each student was required to present a paper, with models and drawings, of the comparative anatomy of some part of the head and neck.

Those who studied under him not only acquired a basic foundation of knowledge but also learned what was even more valuable— the methods of studying and approaching surgical problems of the head and neck.

Though many who studied under him did not appreciate it at the time, it was this valuable training which enabled them to advance the surgery and practice of otolaryngology to its present level.

To those of us who worked under him at the Medical School or in the laboratory, he was a driving taskmaster. He was, above all, a perfectionist; and he demanded the best of everyone while being even more demanding of himself. Many a long night he spent at the Medical School and in the laboratory on anatomical dissection or animal experimentation.

To those who showed an interest in advancing otolaryngology, he gave his fullest support and encouragement. He resented to a marked degree, in his earlier years, the encroachment of private practice on the time which he felt could be better devoted to teaching and research.

Dr. Mosher's students learned more than anatomy. They also learned how to prepare and present papers. He insisted upon clarity. Any kind of "padding" was taboo. "Boil it down" was his constant admonition.

As a showman, in the best sense of the word, he had no equal. His meetings were prepared with meticulous care; nothing was left to chance. As a result such meetings seemed to run themselves. Any

long-winded speaker could expect to be cut short with a "What are your conclusions, Doctor?"

Dr. Mosher's greatest assets, besides his artistic ability, were a lively imagination, an insatiable curiosity, and robust health. He had an uncanny ability to think up problems and to set his students, or himself, to work on them.

Dr. Mosher was a sensitive and lonely man. To those who knew him well, his porcupine exterior was a shield with which to hide his magnanimity. The number of young men to whom he gave a helping hand is legion.

Above all he was devoted and loyal to his ideals, his institutions, and his students. Finally, he was loyal beyond measure to his chosen work. He dedicated his life to it, and the present status of otolaryngology is in no small measure due to his efforts throughout his long and useful life.

Above: with John W. Carmack and Joseph C. Beck, during a session of the Board, Chicago, 1933.

Left: as a Lieut. Colonel in the Army, 1918.

Below: with William V. Mullin, New Orleans, 1932.

III

HARRIS P. MOSHER

A SKETCH IN BLACK AND WHITE

Arthur W. Proetz, M.D.

St. Louis, Mo.

When death came to Harris P. Mosher the face of American Otolaryngology was changed. He was, by common consent, the Dean of the Specialty, an opinion in which he concurred and which he actively fostered. For years he occupied one position of leadership after another, until leadership fit him like a uniform. It dated away back, I have been told, to the early days of his connection with the Department at Harvard, and it grew as his interests widened.

Early in the century, when the specialty, still racked with growing pains, belonged to the few adventurous spirits who had the foresight, the fortitude and the means to make the long pilgrimage to the centers of Europe, personalities were more sharply defined than they are now. It was natural to champion, then to emulate the great teachers of London, Berlin and Vienna. Today there is little impulse to fight any spirited battles over the impersonal streams of technology flowing from a dozen universities for any and all who would drink of their waters.

Nowadays when the germ of every new idea filters out to hundreds of investigators (real and self-styled) almost before it comes alive, there is less rugged contention for a principle, a theory or some elegant electuary than there was during the heyday of Coakley, Delavan, Sluder, Jackson, Casselberry, Swain, Kyle, Ingersoll, Richards, Freer, Bryan, Skillern and, of course, Mosher. Not all of equal scientific stature, perhaps, but vocal and striving like Titans with and against one another, sometimes bitterly, sometimes with a velvet politeness. Mosher was seldom bitter, and not always polite. He had the courage of his convictions, based usually on personal observation, and expressed in cold glittering steel. He loathed pomp and pretentiousness and scorned incompetence in any form. His hobby was anatomy, and he demanded minute details at examinations. Bluffing with him was suicide.

His early uneasiness over the chaotic state of otolaryngological training in this country became acute with the outbreak of the First

World War when he entered the Army Medical Corps as a consultant.

On a gloomy morning in 1917 I was messing about in the small bell-tent which served as the nose-and-throat station of a thirteen-hundred bed tent hospital in the north of France when the flap opened and a visiting American colonel stuck his head inside. It was a British hospital and a strange American officer was a novelty. The conversation which ensued was fairly animated.

That night I asked the C.O. the identity of the American brass.

"Colonel Mosher," he answered.

"Don't know him," I said.

"You will," he replied, "he's by way of being your boss."

Many times since then, in the course of an intimate friendship of thirty years, I have had occasion to reflect that I still did not know him. He was royalty, the Professor, the Chief, the Old Man, the jovial dinner companion and a prodigious crosspatch, all in one, and all at once. When he began chewing his cigar in two one knew that he was either basking in contentment or terribly annoyed.

His conversation was unpredictable. It was apt to be divided between a sort of belligerent jocularity and bitter sarcasm, so that one had to know him well not to be hurt, which resulted in keeping most of his acquaintances and many of his friends at arm's length.

The foreign policy of Theodore Roosevelt, "Speak softly—and carry a big stick" found its antithesis in that of Harris P. Mosher which might well have been paraphrased as "Speak sharply—and carry a small bouquet" for he spent half his time trying to make amends for some pat crack, delivered in jest, which nevertheless had left a sting.

He was never a bystander: one was soon made conscious of his presence in any gathering. His keen and critical mind was constantly analyzing and tearing things apart, trying to improve everything and dropping provocative remarks which sent people scurrying off to take stock of themselves.

He was easily the greatest single influence in raising the standards of otolaryngological teaching in America. The tasks he set himself and his students provided a vivid example for the heads of departments in other universities, and "Mosher's Course" became a by-word in teaching circles. The erudition and familiarity with the literature which came out in his impromptu discussions before the senior societies commanded the respect of his associates, and the terms in which they were expressed discouraged many a loose thinker from rising to his feet.

Above: at a conference of the American Board of Otolaryngology, 1940.

Left: during a meeting of the American Academy, French Lick, Indiana, 1931.

With this background, and his dauntless energy, he was a natural choice as a member of a committee of five appointed to examine candidates for admission to the Academy. This committee which included, besides Mosher, John Ingersoll, Ross Skillern, Thomas Carmody and Joseph Beck (Chairman) first examined in 1921. Four years later when it emerged, somewhat augmented, as the American Board of Otolaryngology Mosher became president and remained president for twenty-two years.

Here it was that his talent for leadership gained full sway, and it was through the Board that his ideas on training for the specialty found their greatest outlet. With the passage of time the increasing requirements of the Board set the pace for postgraduate curricula throughout the country and these were largely the result of his planning. With the devoted help of William Wherry, secretary from 1928 to 1942, he watched over the changing course of teaching in hospitals and medical schools and did his best to guide it in the way he thought it should go.

As an examiner he was a terror, at least in his earlier days. For some reason best known to himself, he would start off with a question which left the candidate limp, and then spend the remainder of the hour rehabilitating him. His grade, afterward, would have surprised no one more than the candidate himself, for he seldom failed a man without some damning evidence from the rest of the Board. His negative vote always came with reluctance, but a thousand times there was satisfaction in his voice as he announced for the record: "The man passes."

Later on his examinations lost some of their fearsomeness, and there were more hints of geniality in his questions, unless the candidate began feeling a little too sure of himself, and then he got a nasty one. Four-flushers had a bad time. The net result was, naturally, that candidates learned their anatomy before stepping onto the mat, and thus a one-man campaign added immeasurably to the basic training in that branch.

He lent his full support to Beck in developing facilities for the study of gross and minute pathology in this country, first by supplying material to students and then by establishing the subject in the examinations at a level which induced the medical schools to offer courses. Physiology was not his bent, and it took argument to get that on the schedule, but soon it came too, along with others of the "basic" sciences.

To him his students, present and past, were the Royal Family. He fought their battles even long after they had left his roof. They worshipped him in return, and regarded his bullying with nostalgia:

"When I was with Mosher, do you know what he said to me?" . . .
"Did I ever tell you what happened the day I came late to class?"
. . . "I only asked him a simple question and . . . "—such was
stock conversation in the lobbies at Academy meetings, and still is,
and it mellows with age. Within his lifetime he became a legend.

He was simple in his personal tastes and cast a cold New England
eye on others less conventional. Once, during a walk, he apologized
to me for stopping in a drug store to buy a nail file. He thought it
was effeminate, but he had forgotten his pocket knife. Another
time, after twitting a colleague for years for wearing a flower in his
lapel, he had a large bouquet brought in to him in the middle of a
public dinner.

His sharp tongue was his worst enemy for it lost, or cooled,
many a friendship for him, which was a pity since the abrupt attack
was often only a mannerism growing out of an intense nature striv-
ing for perfection; to do him justice, he was as critical of himself as
of others. In the somewhat gentler days of his later life one almost
felt that he was playing a rôle; and the character he consciously or
unconsciously portrayed was Harris P. Mosher in his fiery prime,
when he had earned a reputation as a pepper-pot, which now he
rather liked to live up to. After a particularly fine sally one would
often detect a sly side-glance to see whether the shot had been re-
ceived in the spirit in which it was sent. He was merely sparring
for a little stage duel, and was crestfallen when he did not get it.

Idleness was unknown to him. When not occupied with his
teaching, or one of his numerous papers, he was sketching or model-
ing. Traveling about the country, he was never without sketch-
pad and water colors, jotting down what he saw from the train or
a hotel window. He was intent on the artistic and nothing escaped
his eye. Once he asked me to make him a photograph of a mailbox
in a hotel. His talent stood him in good stead after his retirement
from the University and gave him much pleasure.

The life of this remarkable man has left its indelible imprint on
American otolaryngology. His publications will stand as represen-
tative of his day. The ideas set forth in them may be superseded,
and probably will be, but they are honest, and competent, and based
on personal investigation and experience. The inspiration of his
teaching will go on for generations. More intangible than these, but
no less far-reaching, will be the results of numberless small arrange-
ments behind the scenes for the benefit of young men who seldom
suspected they were going on and who sometimes never found out
that the strings which landed them in the fine positions they now
hold were pulled, not from heaven, but from Boston.

CURRENT EVALUATION OF NASOPHARYNGEAL DEFENSE MECHANISMS

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Most otolaryngologists have been trained to an anatomic attitude respecting the defense mechanisms of the upper respiratory tract. Aside from purely mechanical factors, it was for years not considered feasible for most patients to receive treatment based on physiologic lines, until the advent of vaccines and more recently of antibiotics.

While Harris Peyton Mosher was at first intrigued by the anatomy of the ethmoidal labyrinth and the other accessory sinuses, he began the study of regeneration of antral epithelium and (using vital stains) of excised tissue, as long ago as 1931. Proetz and Hilding were already at work on ciliated epithelium; and Larsell and I since 1928 had begun our work on mucosal protection. We made, thereafter, a thorough study of various factors, tending to the belief that the main element in mucosal defense consists in the carpet of mucosal cells lining the nose, nasal cavities, nasopharynx and upper respiratory tract. Herewith, somewhat dogmatically stated, are our conclusions down to the present:

The first line of defense is the covering of mucous secretion on the epithelium, as produced by the goblet cells and glands of the mucous membrane. Inert dust particles and bacteria are removed with the mucus, by ciliary action, helped occasionally by sneezing. Mucous glands and goblet cells are found in the walls of the nasopharynx and also in the lungs.

The content of lysozyme in this mucous coat is variable but constant under normal conditions; and the pH of the nasal secretion is changed materially with the onset of local disease.

Cilia in the nasopharynx and accessory sinuses carry on purposeful activity, keeping a fresh sheet of protective mucous secretion over the epithelial surface. Increased mucous secretion to cope with irritation or infection is an attempt to strengthen the first line of defense, but may produce a blockade in the nasal passages or filling of the accessory sinuses.

From the University of Oregon Medical School.

Virus infestation of bacteria, notably in cases where response to antibiotic medication has been slowed down or lost, may produce severe reaction to bacterial invasion, in which case local use of antibiotics is worthless.

Allergic reaction of respiratory epithelium, with production of local edema and obstructive phenomena due to secondary infection, is controlled by specific drugs acting upon the sympathetic innervation. But local changes due to secondary infection may overshadow the original allergic etiology—and permit the survival of virus strains which will renew their attack upon the cells when bodily resistance is lowered by cold, exposure or unusual mental or physical strain.

The epithelium is thus the actual first defense, but will change with hyperventilation, loss of cilia and trauma, to become stratified squamous in type, with ingrowth of fibrous tissue, which may thicken greatly.

In the paranasal sinuses epithelium is normally simple, thin ciliated columnar, with but few goblet cells and glands. In response to inflammation or blockade, this epithelium swells enormously to a stage which may present in the nasal airway as polypoid protrusions.

The respiratory membranes, resting on a layer of connective tissue, are attached to rigid walls, without muscular movement to force lymph-containing toxic substances along lymphatic channels to help remove noxious elements from the membranes. This slow removal tends to permit accumulation of toxic products, and lymphocytes and neutrophile leukocytes come in by migration from the blood stream. This produces edema and a greatly thickened membrane filled with cells, which begin to phagocytise the bacteria found in the depth of such swollen tissues.

The second line of defense consists of neutrophiles and of "scavenger" cells, the histiocytes, which proliferate or migrate and increase rapidly in number in the early stages of sinus membrane infection. These histiocytes engulf bacteria, supplementing the neutrophile efforts, and may migrate through the epithelium to escape into the sinus cavity. But swelling may close sinus ostia and aggravate the sinus situation; and after a month the histiocytes and leukocytes disappear and are substituted for by plasma cells.

These latter, often in large numbers in "chronic" sinus membranes, are not phagocytic, but are assisted in the task of healing by the histiocytes, which later become fibrocytes and then fibroblasts.

Swelling of sinus membranes, aggravating acute or residual infection, may be produced by vasomotor changes due to reflexes carried

by the autonomic nervous system, often arising from abdominal or pelvic congestion and calling for relief elsewhere than in the nose.

Lymphatic drainage, first to the retropharyngeal and superior cervical lymph nodes, has been traced by vital stains to show that these glandular structures form the third and major line of defense of the body against sinus infection. Localization about such structures may produce abscess; but if the deep superior cervical nodes are overwhelmed, drainage into the cervical lymph ducts may occur, with eventual drainage into the superior vena cava. Such drainage may travel into the right heart and the pulmonary capillary bed— and thus may eventually reach the perivascular and peribronchial lymphatics, which constitute a fourth barrier to systemic invasion by upper respiratory infection.

Fifty years ago Henry Wagner of San Francisco suggested that the natural immunity of the mucous membranes of the respiratory tract rested not on the mechanical protection of mucin, but upon the protoplasmic activity of living body cells, and Harris P. Mosher, artist-anatomist, brilliant surgeon and teacher, dissatisfied with the results of operative procedures, turned to the work of his laboratory for explanations of the healing effects of the cellular linings of the accessory sinuses. May his example not be forgotten!

1020 S. W. TAYLOR ST.

With Ralph Fenton, 1939.   PHOTO WERNER MUELLER

V

A CASE OF MENINGITIS AND BRAIN ABSCESS IN PRE-ANTIBIOTIC DAYS

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This case occurred in 1932 in the beginning of the sulfa era. The patient, H. S., was injured in an automobile accident in 1930, one and a half years before he was first seen by me. He gave a history of fracture of the mandible, upper jaw and of the skull through the frontal sinuses. At the time of his first visit there was a large depressed area over the left frontal sinus with a draining fistula over the left inner canthus, complete ptosis of the left upper eyelid and failure of the left eye to rotate upward. His nasal septum was markedly thickened and deviated to the right, obstructing both middle meatuses, but especially that on the right. The middle turbinates, what could be seen of them, appeared to be hyperplastic and there was complete anosmia. There had been profuse purulent discharge from the left nostril until two months previously. Vision was normal in both eyes.

On January 18, 1933, a submucous resection was done to improve nasal drainage as a preliminary to frontal sinus surgery. This was without incident and the patient was discharged to return shortly for the further surgical procedures necessary. On February 25, 1933, operation having been postponed for one week because of glycosuria, both frontal sinuses were opened externally. The mucosa was found to be extremely hyperplastic and the sinus cavities full of pus. A considerable amount of gravel was removed from the external outer angle of a very large left frontal sinus. The anterior wall of this left frontal sinus, was depressed and adherent to the inner table and there were large infected supraorbital ethmoid cells on each side as well as a cell in the intersinus septum. The left supraorbital ridge had been fractured and was depressed. The previously existing fistulous tract above the left inner canthus was closed by swinging over a plastic flap from the right forehead. After a thorough cleansing of both frontal sinuses removal of the intersinus septum and enlarging the nasofrontal ducts, further surgery for the ptosed upper eyelid and the superior eye muscles was deferred on the advice of Dr. E. B. Spaeth, until all infection had cleared up. The patient made an un-

eventful recovery and was discharged with an external depressed deformity over the frontal sinuses, but no evidence of infection. He was not seen again until March 20, 1937, four years later.

On this date he was brought in to the Graduate Hospital of the University of Pennsylvania from his home on the New Jersey coast, 80 miles away, at 3 a.m. He was semi-comatose, but could be aroused. Thirty-six hours previously he had had intense headache and projectile vomiting. He was markedly opisthotonic, with turbid (890 cells) spinal fluid under pressure. Prontylin (sulfanilamid) had recently become available and was under investigation, so, with the advice of Dr. Sergent Pepper, of the medical staff, he was given the maximum dosage orally. This resulted in three days in a remarkable abatement of all signs and symptoms; fever which had reached 104, dropping to 100, normal pulse, no neurological signs or headache. He, however, developed some cyanosis and crystaluria appeared, so that the administration of the sulfonamides was stopped, whereupon he promply returned to his former condition. These alternations of severe symptoms, remission under the administration of the drug, cyanosis, stoppage of medication and return of symptoms, continued with gradually increasing improvement for three weeks, when the sulfonamide was finally discontinued and there was no return of meningeal symptoms.

Before discharge on April 20, 22 days after his original attack a final careful check-up revealed a polyp high up in the right spheno-ethmoidal recess. This was removed and found to be one centimeter in diameter, hard and fibrous with a thin pedicle. After its removal there was disclosed a granulating surface in the region from which it sprang. A probe passed into this area met with no resistance and evidently entered the brain through a fistulous tract several centi-meters deep; at this point resistance comparable to that felt in probing an encapsulated brain abscess was apparent. The probe was with-drawn and was followed by a stream of pus, so that undoubtedly a brain abscess had been entered through a pre-existing stalk. Since roentgenological studies had shown involvement of both sphenoid sinuses, the right one was now opened and found to contain pus and degenerated mucous membrane. The upper portion of the face of the sphenoid was continuous with the granulating area mentioned above. Here were found loosened plates of necrotic bone with moth-eaten edges, which were carefully removed, uncovering the dura to about one-half the size of a ten cent piece. The left sphenoid sinus was then drained. At this time the patient's wife informed me that ever since the original automobile accident he had had an intermittent leakage of clear fluid from the right nostril—evidently a cerebrospinal

rhinorrhea.   He had not thought it worth while to report this at previous history taking.

Other than a headache for a few hours, there was no reaction, but for several days there was a profuse drainage of purulent material into the nasopharynx.

It would seem that the feeding source of the patient's meningitis had undoubtedly been in the right-sided brain abscess and the necrotic bone in the sphenoethmoidal recess.

The subsequent course was uneventful.   After a week, drainage had largely ceased and he was discharged in an apparently normal state of health.   He was to return later for surgery of the left orbit. He was next seen in August of the same year, there having been no change in his condition.   There had been no headache.   He reported from time to time during the next fifteen years that he had been in perfect health, but he never returned for corrective surgery of the left eye.

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VI

SOMATOPSYCHIC AND PSYCHOSOMATIC FACTORS IN TINNITUS, DEAFNESS AND VERTIGO

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AND

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The inner ear is an ingrowth of the ectoderm or outer skin of the primitive embryo. Even from its beginning in these fish-like stages it is a protective device, and as it develops it becomes more and more efficient and delicate. No electronic device can equal the cochlea for sensitivity, precise response, frequency or intensity range, not to mention smallness. The cochlea can be stimulated by the impact of anything larger in diameter than that of the hydrogen atom.[1] It is estimated that this is the amplitude for threshold stimulus at 1000 cps. It is claimed that in a sound-proof room the ear can sense the noise made by the jostling of the molecules of the air. The modern hearing aid, the decibel meter, or even the finest high fidelity radios are not in the same class with the cochlea.

As for the vestibular system, the utricle and the semicircular canals, they function more rapidly and precisely than machines, even than diminutive automatic pilots which moreover measure perhaps 10 x 10 x 15 inches. Considering the delicacy of the labyrinth it is remarkable that it so rarely gets out of order and that when it is damaged it does not always go completely out of order. However, it is well known and not remarkable that the smallest change in its circulation can produce profound symptoms since we know that the labyrinth is connected by reflex mechanisms to every muscle in the body and to some of the viscera. The inner ear, being designed for normal physiological stimuli, easily temporarily gets out of order when subjected to violent stimuli such as those encountered in modern air plane flight or during violent emotional stresses so often found in modern life. Even so there are many factors which contribute to its ruggedness. This probably is a hereditary trait. Unquestionably some people have tougher ears than others. We do not really know why and we shall have to accept it just as we do big feet or height or blue eyes.

There is more to this heredity story. Even when we know that certain individuals are hereditarily predisposed, or more susceptible to certain diseases and symptoms than other people, is there anything we can do about it? Can we prevent or alleviate these troubles before they cause symptoms? There is some evidence that we can. How do we go about it?

We study the somatotypes i.e., the body types and emotional make-ups, and then we try to interrelate the emotional episodes with the appearances or exacerbations of the disease in each type.[2] We find that we can do so to a certain extent. With further work and study we should be able to do even more than at present. It is in this field that our laboratories and our clinical researchers are now working. Some preliminary results may be reported as follows:

Ménière's disease, sudden deafness and otosclerosis patients can frequently, and with high statistical validity, be classified into certain body types. Furthermore the closer to certain prototypes these cases are the more severe their symptoms tend to be, and on smaller provocation. Our observations allow us to assume that given a heavy-set mesomorphic type with Ménière's disease, unless we change the patient's life pattern, he will become worse, and even develop the disorder bilaterally, and with exacerbations in symptoms and extensions of the lesions in the inner ear.

To be sure, the tall, leaner, types may develop symptoms of the disease but as a rule this occurs only under severe provocation, such as after the death of a loved one, the defalcation of a trusted partner, the frustration occasioned by business difficulties or the care of the incurably ill.[3] With these nonmesomorphic patients we usually need not be so much concerned as with the squat, muscular types.

The more emotionally disturbed the patient and the closer he is to a classical disease somatype the more assistance he will need from an understanding physician. During each office visit we must stress one or more of the following: The necessity of lessening high pressure, increasing humility, eliminating or cutting down tobacco, caffeine, aspirin and other ototoxic drugs, and in fact all drugs, easing frustrating work and dwelling on the past, avoiding fighting with one's fellow men actually or in thought, and in general attaining more equanimity. Note that we have not suggested that man or woman should retire or do nothing. For a fairly normal man or woman this is often a catastrophic thing to do. The emphasis should be on taking some of the straws off the camel's back. Fortunately, management and treatment along these lines is also good for the general health. One regret of the modern physician is that his religious

confreres seem less able than in the past to persuade people to lead a sensible life. Nowadays even the Sabbath is not really a day of rest. Making it more relaxing will often aid a patient in relieving his tinnitus and his vertigo.

Too many otologists, like other physicians and surgeons, tend to employ medicine or surgery, or both together, as complete remedies. In truth neither alone, or both together, should be considered complete because both supplement, they do not replace the inherent reparative processes of the body. They aid them, but other modalities also aid in the alleviation of a patient's sickness.

All disorders and diseases of the body tissues, or body functions, are complex and regularly occasion in great or small degree both higher center, as well as lower center, neural responses. As we have indicated before, it may be because the ear is so sensitive that we are more likely to derive symptoms from it than from other sense organs.

Central nervous system responses, when they are detectable, for want of a better name are commonly called "emotional." We do not like this word because it has certain false implications. The nervous system may respond on a reflex basis without any "emotional" component whatsoever. In many conditioned responses this is the role.

Whenever one or more of the eleven senses are stimulated the autonomic neural mechanisms respond because a person is dependent upon his cognizance of sensations to orient him and protect him against adverse factors in his environment, external and internal. Therefore, any abnormalities of the sense of touch, vibration (hearing), sight, equilibrium, progression, smell, etc., in the higher animals at least are met with efforts to enjoy, act upon, overcome, adjust to or circumvent such sensations depending upon their so-called physiological or pathophysiological effects, and especially upon their distortion. Permanent undersirable results occur only with excessive concern, stress, anxiety, fear, alarm or anger, etc., all primarily essentially defense reactions. It is only when the nervous system is overworked (overburdened) that pathological symptoms and diseases develop, and no two people have the same threshold of resistance to potentially damaging stimulations.

Whenever the physician is unable to diagnose disease in some organ or mechanism, he is of late liable to designate the symptoms as "psychosomatic;" not a very good word but one in common use at the moment. It implies that the symptoms or disease processes are caused not primarily in the tissues involved, but are caused by mental processes in psychologically predisposed people, who are constitutionally hypersensitive to certain precipitating factors in the

organ affected.   A diagnosis of psychosomatic disease is too often a subterfuge to save face.   The designation "psychosomatic" or "emotional" is an oversimplification, because every disorder or disease affects not only the psyche but also the soma, at least to some extent. "Somatopsychic" is often what the physician really means by the term.   "Somatopsychic" implies that the patient is emotionally upset by his primary disorder or disease in some tissue or organ.   Psychosomatic means that a real disease process has been set in motion by an overloading and breaking down of body defense mechanisms.   This is usually an unconscious reflex affair, but whether it is high or low in the spinal cord, brain stem or cortex it is often possible to uncover a trigger mechanism for it.   In any case, finding out that the disease is psychosomatic or has psychosomatic features should not relieve the physician of the necessity of doing something about it.   But we repeat, no one mode of treatment, whether it be psychotherapy, surgery, medicine or other, should be used exclusively.   Medicine and surgery should be employed to hasten recovery of the patient, not alone the symptoms of the disease from which he appears to be suffering.   Whether we like it or not, it is a fact that our patients often derive as much, or more, benefit from our counseling and understanding than from our medication or operation.   The point we are belaboring is that in using medicine and surgery properly, to aid nature in overcoming disease in certain organs, we should not neglect man as a whole individual.   We must instill in our patients a philosophy of sickness, yes, and a philosophy of life, of living and even of dying, all inevitably related to a mature and satisfactory sojourn on this earth. About most things in medicine one must be factual.   It is the doctor's duty to teach his patients to be factual and even about some things to be fatalistic.

To be more specific, let us now discuss tinnitus aurium.   About 85 per cent of all of our adult patients have complained of or have been conscious of head noises of some kind or other.   How do we go about evaluating them and determining the factors which cause them and often make them seem unbearable?

What do we do to supplement our medication and surgical treatment?   Without some previous knowledge of the disordered sensations, without some conception of the symptoms, no disease of the tissues is conjured up simply by thinking about it, but who among us has not had symptoms referable to many organs of the body, some pains or sensations which in apparent health appear normal?   Everyone has experienced at least transitory pains of various kinds, not only definitely during waking hours but indefinitely during sleep, and from both known and unknown causes.   Who has not experienced

tinnitus, unsteadiness or dizziness or real vertigo and maybe nausea, and even on occasion vomiting (not necessarily following alcoholic excesses).

As for deafness, no one has escaped some difficulty in hearing, transitory or for a period of time, due to an actual ear disease, to impacted cerumen, to covering the ears, or from loud noise or acoustic trauma, etc. Similar comments could be made of all of the sensations and their organs or origin. In hypersensitive people such sensations (or illusions of sensations) can be, and often are exaggerated, sometimes into a state of fear verging on hysteria; in some into a state of manifest and marked hysteria. There is a point beyond which legitimate concern blows up into a true anxiety neurosis.

At this point a word about iatrogenic disorders. Some physician's in their attitudes appear to overstress the patient's symptoms and instill additional alarm and fear, or they may understress them to such an extent that the patient suspects he is being treated like a child or a moron, that the doctor is afraid to tell him the whole truth about himself. Cancer, total deafness, chronic invalidism, brain tumor or loss of sanity may then loom large on the horizon. The result may be increased concern, increased anxiety, increased fear and stress, and increased suffering, and most important of all, an increase of the symptoms and even of the diseases or disorders connected with them. The result can be called "iatrogenetic," an imposed overconcern and exaggeration of symptoms.

Children, before puberty, seldom mention tinnitus or monaural deafness, and even binaural hearing losses may be taken in stride. Children seldom mention mild or unilateral deafness, or any other moderately severe symptom, unless it is associated with pain. This is one of the reasons why children can be the more regularly restored to satisfactory status; they can be rehabilitated more readily than adults, unless their elders, especially their parents, are neurologically unstable and handle both themselves and their child badly.

The prime requisite for successful treatment is to explain in simple words the reason for the symptoms and then to make the patient understand how severe they really are. For example, the real loudness of the head noises, and the real loss in capacity to hear, can be readily demonstrated. If a patient is shown that the deafness or the head noises should not really be unbearable, that they are not going to result in insanity or total deafness, you have made a good start. Then with full co-operation the patient can be helped to lessen, or to tolerate his symptoms. This will be the result unless he fails to co-operate or the otologist fails to maintain interest. A satisfactory

result can be attained even with severe vertigo if we use sufficient patience and sympathy (not too much sympathy).

Of course proper medication is of service but it will not suffice alone. Medicines alone by their side effects may even increase the emotional hypersensitiveness and cause severe undesirable consequences, such as indigestion, malnutrition, neurovascular repercussions, and diminished well-being.

Tinnitus, as well as vertigo, may be temporarily smothered by many narcotics, sedatives, and a few drugs, notably procaine, but relief will be transitory unless the emotional factors are also relieved or removed.[4]

Exaggeration of symptoms of ear diseases may exist long before any iatrogenetic influence of the physician. Friends, or family, may be the principal offenders. Many people love to describe their troubles in detail. The worst offenders come in repeatedly with long, long-hand or typed lists of questions and symptoms. It is well to remember that sometimes before these patients consult a doctor they have been neurotic and that their present symptoms are grafted upon this neurotic background of chronic anxiety which ordinarily they may conceal except during phases of aggravation.

In some people the neurosis appears to be a compelling urge to suffer, to arouse sympathy and concern. Should we estimate their exaggerated behavior as genuine, or as largely a symbolic expression and substitute, rationalized for unacceptable threats to the personality, some symbolized emotional reaction representing other greater though undisclosed fears or concerns? Paradoxically it may be both and thus eventuate into an ever increasing vicious cycle.

Ask the patient, "How would you feel if I discovered you did have a very serious disease?" Notice his facial expression.

Evasiveness: Many will admit that they would be greatly relieved, but you can detect they are not—they harbor a persistent obsession that more is wrong with their ears than anyone knows. The normal person accepts facts and usually courageously.

Ineffectiveness of reassurance: a seeming disappointment in negative reports—a history of shopping about, and consulting many physicians, is typical of emotional instability.

Never give a patient a list of the disagreeable possibilities. Usually it is well to delay telling these patients your complete diagnosis. Tell them they must be studied; make a definite date for further tests; strengthen confidence by listening to their complaints. Be hu-

man and soon they may, and often will, ease up on their emoting and reveal their real troubles and fears.

We will spare you statistics. But we can state that treating people with tinnitus, many forms of dizziness, and of deafness, with the approach outlined, has with few exceptions been satisfactory in that the patients so treated have been wholly or greatly relieved of their symptoms, and returned to what could be called a more normal existence and happier life, symptoms or no symptoms.

How do we examine people who suffer from tinnitus?

1.   Listen attentively to their story and obtain a complete personal history, particularly concerning the symptoms of the present and past episodes possibly related to emotional strains at home and in the business world. Ascertain habits as to smoking, coffee, tea, alcohol and drugs; also as to any exposure to industrial or excessive noise, at any time.

2.   Obtain a real family history (comprehensive) not only concerning disease, but also the personality of siblings and immediate ancestors.

3.   Examine carefully, not only otolaryngologically but as a physician. Be sure to get a history covering endocrines, heart, blood pressure, lungs, digestion, genitourinary tract and neurological signs. If possible obtain a complete general physical examination, include blood pressure, blood count, basal metabolic rate sedimentation rate and urine examination, etc. If you do not, some day you will be caught flat-footed with an arteriosclerosis, a leukemia or anemia causing the tinnitus, or with some thyroid disease causing or at least at the bottom of the symptoms.

4.   Measure the hearing capacity.

5.   Measure the loudness of the tinnitus, and its approximate frequency, or frequency band.

6.   Estimate the degrees of any vertigo, nausea, vomiting, present or past. Make a careful and complete diagnosis, not only as to the location of the lesions, but as to the pathology, pathophysiology, etiology and prognosis.

7.   Examine carefully for spontaneous and postural or positional nystagmus and determine by rotation, caloric or optokinetic testing the degrees of abnormal reactions obtained. You may be dealing with a brain tumor. Do not at first use the severe tests because the response may be misinterpreted on account of the "recruitment" of sensation or because of hypersensitivity of the apparatus tested.

8.   Tell the patient the results of your examination in a clear, confident, reassuring manner, and outline (only outline) what treatment and management is indicated, if any, and what medical treatment and what surgical measures are not indicated.

9.   Explain simply and briefly the basic types of tinnitus and their pathophysiology. Be sure the patient is made to understand that intrinsic or nonvibratory tinnitus is not a real sound but merely a sensation of sound, not a real sound but an illusion of sound, as itching can be an illusion of touch to the skin, or a flutter of light can be an illusion of light when the eyeball is tapped.[5]   Impress on him that it is *not a delusion,* and that unless he was crazy before he saw you he probably will not be crazy afterwards.

Then give the patient your treatment of choice at the moment. We use benadryl, atropine, nitroglycerine or scopolamine for acute attacks; nicotinic acid, potassium iodide, and perhaps intravenous procaine for the long pull; dramamine or small doses of benadryl for mild dizziness—all laced in with strong reassurance and insistence on developing a new philosophy of life, which latter will make subsequent attacks less likely.[6, 7]   In very severe Ménière's disease it may be necessary to undertake procedures like the Day or Cawthorne operations. It may even be indicated to use streptomycin, but this is necessary only in certain instances where the physician can put his finger on the real cause of the tinnitus, nerve deafness and vertigo: namely, at least in part, and in fact usually largely, the patient's repercussions from his way of life. If he can be taught to become less frustrated, less aggravated by his business and his family and friends, the battle is won.

Most people with the symptoms under discussion are old enough so that their limitations can be fairly appraised. They should be taught to accept some of their limitations; they should be cautioned against doing so many things. When they become adjusted they will be healthier and happier, and you have won.[8] Somehow the tinnitus and the vertigo will have disappeared. If one can teach people equanimity early enough sometimes even the hearing may improve, whereas usually the hearing remains stable or slowly deteriorates. In any event the patient must be taught to live with himself and his infirmities, and be grateful that neither is worse. If he persists in resisting advice and exhausts your patience, he had better consult one more skilled and rugged than you as his doctor. We trust he will have difficulty in finding such an one.

140 EAST 54TH STREET.

REFERENCES

1. Davis, H.: Hearing and Deafness, Murray Hill Books, New York, 1947, p. 27.

2. Damon, A., Fowler, E. P., Jr., Traube, S. and Sheldon, W. H.: Constitutional Factors in Ménière's Disease and Otosclerosis, Trans. Am. Acad. Ophth. and Otolaryng. (In press).

3. Fowler, E. P., Jr. and Zeckel, A.: Psychosomatic Aspects of Ménière's Disease, J. A. M. A. 128:1165-68, 1952.

Fowler, E. P., and Zeckel, A.: Psychophysiological Factors in Ménière's Disease, Psychosomatic Medicine 15:2, 1953.

4. Fowler, E. P., Jr.: Intravenous Procaine in the Treatment of Ménière's Disease, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 62:1186-1201, 1953.

5. Fowler, E. P.: Nonvibratory Tinnitus, Factors Underlying Subaudible and Audible Tinnitus, Trans. Am. L. R. & O. Soc., 1947.

6. Fowler, E. P.: The Emotional Factor in Tinnitus Aurium, Laryngoscope 58:145-54, 1948.

7. Fowler, E. P.: Tinnitus in the Light of Recent Research, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 50:139, 1941.

8. Fowler, E. P.: Tinnitus, Vertigo and Deafness (Ménière's Disease and Ménièriform Disorders), Practice of Otolaryngology, Ed. George M. Coates, et al (In press).

BILATERAL ABDUCTOR PARALYSIS OF THE LARYNX

RESULTS OF TREATMENT BY MODIFIED KING OPERATION

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Bilateral paralysis of the larynx occurring as a complication of thyroidectomy practically always is of the abductor type. It is due to injury to subdivisions of both inferior or recurrent laryngeal nerves supplying the posterior cricoarytenoid muscles. The immediate result is marked adduction of both arytenoids. Dyspnea often is urgent and immediate tracheotomy may be required. The voice commonly is hoarse; the patient may become aphonic. When the larynx is examined within 24 or 48 hours one finds the arytenoids and vocal cords adducted, the arytenoids being erect as though in the phonatory position. There commonly is some relaxation of the vocal cords. Later the tension improves and the voice assumes a normal sound in the conversational range. Dyspnea, particularly on exertion, breathlessness and stridor persist and may increase with improvement of cordal tension.

The prognosis for restoration of function is not good. In not more than three per cent of cases is there unilateral restoration of normal movement; in none is there restoration of normal movement bilaterally.

While bilateral paralysis of the larynx complicating thyroidectomy has been long recognized the treatment has been varied. The earlier surgical procedures consisted of removal of a portion of a vocal cord either by direct laryngoscopy or thyrotomy. The immediate results were satisfactory but after fibrosis occurred one frequently was confronted with cicatricial as well as paralytic stenosis. During the 20's there were many unsuccessful attempts to resuture the severed ends of the recurrent nerve or to anastamose the distal segment of the recurrent nerve with a motor branch. Inability to identify the distal segment of the nerve was responsible for many of the failures.

The operation described by King[1] in 1939 represented a rational extralaryngeal approach towards increasing the laryngeal airway; however, there was no restoration of movement to either arytenoid. In early reports it was stated that the transposed omohyoid muscle

imparted movement to the attached arytenoid. This never was sub-stantiated. Motion pictures of the larynx made at mirror laryngo-scopy before and after operation in a number of cases revealed con-clusively that the arytenoid and vocal cord on the operated side remained immobile; the only movement noted during phonation occurred on the unoperated side.[2] This observation was made by others and it now is generally accepted that the success of this plan of treatment is dependent on free mobilization of an arytenoid and its lateral fixation by some form of suture material. The role of the omohyoid muscle is unimportant and undependable for purposes of fixation or affording mobility. One of the great advantages of the modified King procedure is an absence of postoperative cicatricial changes within the larynx. An adequate airway once secured by this method therefore can be considered as practically permanent. A number of modifications of this operation have been introduced but the fundamental principle, namely mobilization and lateral fixation of an arytenoid, remains unchanged.

My experience in the surgical treatment of bilateral abductor paralysis of the larynx following thyroidectomy has been confined to the use of a modification of King's technique and includes a series of 103 cases. An additional nine cases of stenosis of the larynx ap-parently from bilateral abductor paralysis due to other causes also were treated by this method.

Sex and Age. There were 98 females and 14 males. The pre-ponderance of thyroidectomy in females explains the disproportion in sex. The ages varied from 14 to 76 years. Over one-half of the patients were more than 35 years of age.

Etiology. In 103 patients the paralysis followed one or more thyroidectomies. In 7 of these, bilateral paralysis occurred after a third and in one after a fourth thyroidectomy. There were no data available regarding the condition of the larynx prior to the last oper-ative procedure but in every case there was a history of dysphonia for a variable period. It is very probable that all had a unilateral paralysis following the previous operations.

Duration of Paralysis. In 51 cases the paralysis was of less than 5 years duration. In 24, it was from 5 to 10 years, in 19, from 10 to 20 years and in 8 it was over 20 years, the longest being 33 years. In the recent cases operation was deferred for at least six months after occurrence of the paralysis on the assumption that if function was restored spontaneously it would occur within that interval.

Tracheotomy had been performed either during or sometime after thyroidectomy in 33 cases. One patient wore a cannula for 16 years,

another for 17 years, the longest period being 23 years. In 7 cases, tracheotomy was performed on two or more occasions; in one, it was done four times.

Appearance of Larynx. The appearance of the larynx was practically identical in all cases. The arytenoids were in a phonatory position with the vocal cords adducted so that the glottis was not more than 1 to 2 mm in width. In some there was definite thickening of the cords, due either to thyroid deficiency or chronic inflammation. Stridor, breathlessness and dyspnea of varying degrees were common symptoms. The speaking voice was clear but breathlessness often interfered with normal conversation.

The duration of the paralysis apparently did not alter the appearance of the vocal cords. The so-called "cadaveric position" was not observed in a single case. This is not remarkable since such a finding would imply that the trunks of both recurrent laryngeal nerves had been interrupted. There also would be an absence of dyspnea.

Treatment. In discussing the surgical treatment reference will be made only to the surgical procedures carried out in this series of cases.

It is imperative that a careful preoperative medical survey is carried out. In addition to thyroid deficiency there often is calcium imbalance, myocardial disease and chronic bronchitis. In several cases there was present a definite bronchiectasis.

Technique. In patients wearing a tracheal cannula one may insert an intratracheal tube preoperatively through the tracheotomy fistula, thus keeping the anesthetist out of the field of operation and diminishing the dangers of wound contamination. If no tracheotomy has been performed, one either can insert an intratracheal airway through the larynx preoperatively and perform tracheotomy at the termination of the operation or tracheotomy may be performed preoperatively and an airway inserted through the tracheal stoma. While placement of an airway in the larynx often facilitates localization of the arytenoid, it increases the likelihood of injury to the mucosa when separating the aryteniod and also interferes with performing direct laryngoscopy to determine the width of the glottic chink at the time of operation.

While the choice of preoperative medication and anesthesia must be left to the individual operator, it is, nevertheless, imperative that no preliminary sedation or general anesthesia be employed in a dyspneic patient who has not been tracheotomized. Tracheotomy should

always be done under local anesthesia without sedation. Local anesthesia has been employed in several cases of mobilization but has not been satisfactory and either inhalation anesthesia or sodium pentothal are preferable.

A horizontal incision two inches in length and conforming to the skin creases of the neck is made over the wing of the thyroid so that it crosses the posterior border of the cartilage slightly below its middle. The omohyoid muscle may be retracted or incised. The posterior border of the thyroid cartilage is identified and the inferior constrictor muscle is incised along its attachment to the cartilage. The palpating finger should now identify the signet of the cricoid cartilage along its upper margin. As a rule the arytenoid cannot be palpated due to its forward displacement.

The pharyngeal mucosa is separated for a short distance from the signet of the cricoid cartilage towards the midline and the posterior cricoarytenoid muscle is identified and incised along its attachment to the muscular process. As a rule there is little difficulty in identifying and finding the muscular process of the arytenoid. I have found a small pointed hook valuable to secure the arytenoid during these manipulations. In order to secure satisfactory mobilization it is well to separate all of the posterior cricoarytenoid muscle from the muscular process, to incise the fibres of the arytenoideus muscle as much as one can along the body of the arytenoid and then to pull the arytenoid backward to separate some fibres of the lateral cricoarytenoid muscle. The cricoarytenoid joint then is opened and its attachments are incised. Care should be exercised to avoid injury to the laryngeal mucosa, particularly when separating the mesial attachment of the capsule. A blunt elevator has been satisfactory for this procedure.

Before proceeding with the placement of the suture it is necessary to assure oneself that the arytenoid is freely mobilized. It should be possible with the aid of the pointed hook to draw the arytenoid laterally and posteriorly so that it can be brought in contact with the posterior border of the thyroid cartilage.

Lateral fixation is accomplished by securing the arytenoid to the posterior border of the thyroid cartilage. For this purpose a full curved needle with a braided No. 0 silk suture is passed around and through the cartilage and secured through a hole made with a dental burr near the posterior border of the thyroid cartilage. This gives better fixation than if fascia or muscle is used. The suture should be placed around the arytenoid near its base, beneath the mucosa passing the needle from within outward. Occasionally it is difficult

to place the suture in proper position about the arytenoid since this cartilage is pyramidal in shape and the mucosa along its inner surface is in close relation to the perichondrium. In difficult cases the mucosa overlying the posterior and particularly over a part of the inner aspect of the arytenoid should be separated. Perforation of the overlying mucosa may lead to the formation of a granuloma or to infection of the wound. While it is desirable to pass the needle around the cartilage I am sure that in many instances it is passed through a part of the cartilage along its inner aspect.

When drilling the hole for the suture one should try to maintain the arytenoid on its normal horizontal plane or slightly higher. If the opening is made higher so that the arytenoid is elevated when secured with the suture the airway will be correspondingly increased, but this also will produce increased impairment of the voice. The inferior constrictor muscle is then repaired and the wound closed. A rubber tissue drain should be left in situ 48 hours if the mucosa has been injured. A nasal feeding tube may be employed for several days.

Variations in Technique. It is often difficult to secure an adequate exposure of the arytenoid cartilage. Retraction of the wing of the thyroid may be facilitated by separation of the cricothyroid articulation or by clipping the inferior cornua of the thyroid cartilage with shears. Counterpressure on the posterior border of the opposite wing of the thyroid cartilage often gives a better exposure.

Ordinarily if the arytenoid is brought in contact with the inner aspect of the thyroid cartilage along its posterior border the glottic opening will be adequate. In two cases of paralysis occurring in men, it seemed desirable to provide a wider glottis than is secured by this method and a small notch was made in the posterior border of the thyroid cartilage so that the arytenoid could be hinged around it. In an occasional case a second suture has been placed about the arytenoid to aid in its lateral displacement.

It is difficult to be absolutely certain even by a direct laryngoscopy that the airway will be adequate. The occurrence of submucosal edema or hemorrhage during the operation or an active pharyngeal reflex in a lightly anesthetized patient may be misleading. An adolescent laryngoscope is preferable to the anterior commissure type. In the average female the glottic chink should be not less than 5 mm in width posteriorly to insure an adequate airway. In the male a wider airway is desirable.

End Results. It is impossible to restore normal laryngeal function in a case of bilateral abductor paralysis. To improve the airway

one must widen the glottis and this obviously will detract from the patient's ability to phonate clearly. One therefore can state positively that the wider the glottis is, the poorer the voice will be. It has been observed that the operated side becomes fixed, and any movement that occurs is on the unoperated side; therefore, while one may operate on both arytnoids a unilateral operation is preferable from the stand-point of phonation. Better results also will be obtained if the ary-tenoid is retracted laterally without rotating it so that a tense straight-edged vocal cord will be secured.

I had previously believed that the adduction which is observed on the unoperated side could be explained by the action of the crico-thyroid muscle which, although a tensor, also exerts some adductor action on the vocal cord. When the cricoid cartilage is drawn to-wards the thyroid cartilage by the cricothyroid muscle during phona-tion there apparently is some tilting inward of the corresponding arytenoid, which probably is a passive action. There is considerable question whether the action of the cricothyroid muscle is of great significance in posticus paralysis. The arytenoid is tilted forward because it lacks normal fixation and this interferes with the tensor action of the cricothyroid muscle. It seems reasonable that the un-opposed lateral cricoarytenoid muscle may be responsible for slight adductor action on the nonoperated side. I believe that many patients use the extrinsic muscles which aid in narrowing the airway during phonation. In addition one must take into account that there is a change in the upper orifice of the larynx, for the epiglottis usually is tilted backward and on phonation this is increased, contrary to find-ings in the normal larynx.

Voice changes. In none of my operated cases has there been any material improvement in the voice. In practically all of the cases of paralysis following thyroidectomy the voice was quite clear before operation although there was breathlessness. In a majority the voice has been reasonably good following mobilization. In two the air-way was very wide and in these the voice was a loud whisper.

In one, I attempted to narrow the glottic chink by inserting a portion of costal cartilage beneath the perichondrium with but slight improvement. Patients who have worn a tracheal cannula for re-lief of dyspnea or have lived a precarious existence because of short-ness of breath invariably are extremely grateful when the airway is increased. I have never observed a single instance in which the pa-tient regretted having had the operation performed in spite of the voice impairment.

Statistical Data. To date I have performed a modified King operation on 103 cases of bilateral abductor paralysis following thy-

roidectomy and on 9 due to other causes. Of the postthyroidectomy group it was necessary to operate on the opposite arytenoid to secure an adequate airway in eight instances. In the first three treated by the original King technique the omohyoid was transposed with no obvious improvement. The procedure was modified and an adequate airway was secured after a second operation. In a fourth case there was persistent edema of the arytenoid apparently due to chondritis; decannulation was possible only after operating on the opposite arytenoid. Of the remaining four cases two were operated on within six weeks as the airway was found inadequate; a third became dyspneic 15 months after the first operation due to narrowing of the airway necessitating a second operation. The eighth case, previously operated upon in 1940, returned 14 years later with recurrence of dyspnea. The airway was found narrowed but a satisfactory result was secured by operating on the opposite arytenoid.

The question arises whether one might attempt to again mobilize the arytenoid previously operated on. I attempted this on one occasion and found it impossible to separate the arytenoid from the cricoid due to fibrotic and possibly osseous changes.

Decannulation Following Operation. In the earlier cases decannulation was delayed a number of weeks. Now, however, it is carried out more promptly as there has been opportunity to observe the postoperative reactions. This also may be due to the employment of antibiotics or to more careful handling of tissues. In 28 of the last 30 patients treated decannulation was carried out within two weeks, the earliest being five days postoperatively. In one case the tube was not removed until after three months. This patient developed a small granulation in the posterior commissure on the operated side that suggested a contact ulcer although it probably was the result of trauma to the mucosa. Removal of the granulation was followed by complete recovery and decannulation with a reasonably good voice. In a second case decannulation was delayed seven months. The patient had previously been irradiated and bilateral laryngeal edema delayed removal of the cannula.

In three cases decannulation has not been done. In one, an early case operated upon when the omohyoid was used, the patient, a teacher, preferred to continue with a valved cannula. Another patient, operated on by me in a distant city has an inadequate airway and needs an operation on the opposite side. The third case, a patient with basal bronchiectasis will have an adequate airway when the pulmonary infection has been cleared. She had a unilateral lobectomy and requires additional surgical treatment.

Wound Infection. This occurred in five of the earlier cases when the laryngeal mucosa was perforated. In one this was protracted but an ultimate satisfactory result was secured in all cases.

Mortality. There were no deaths as a result of this procedure.

While a majority of the cases of bilateral posticus paralysis occur during thyroidectomy, other etiological factors may be responsible. I have treated nine cases in this group. In three, there were gunshot or shrapnel wounds of the neck, two had pulmonary tuberculosis and in four the cause was unknown. All had been dyspneic or had worn a tracheotomy tube for more than one year. On the basis of a direct laryngoscopic examination it was believed that at least one arytenoid could be moved passively with laryngeal forceps.

There were three failures in this group. These included two cases complicating pulmonary tuberculosis and one due to trauma. A laryngostomy was later done in two and very definite cicatricial changes were found in the interarytenoid area. This accounted for the failure of the primary procedure and undoubtedly was not recognized at the time of the laryngoscopy. In my opinion it is often difficult to differentiate between bilateral abductor paralysis of the larynx and bilateral fixation of the cricoarytenoid joints by direct laryngoscopy performed under local anesthesia.

SUMMARY

A group of 103 patients with bilateral abductor paralysis of the larynx complicating thyroidectomy and nine due to other causes is presented. The surgical treatment, postoperative complications and end results are discussed.

Satisfactory results were secured by fixing the arytenoid to the corresponding wing of the thyroid cartilage with a nonabsorbable suture after free mobilization. In 11 patients it was not possible to complete decannulation after the first operation. Of four failures occurring when transposition of the omohyoid muscle was done three were decannulated after a second operation. Four additional patients required an operation on the opposite side. In two this was done within six weeks. Two others developed dyspnea 15 months and 14 years respectively after the initial procedure; complete relief followed a second operation. Three have not been decannulated. In two, a second operation will be necessary and in the third, decannulation can be completed as soon as the pulmonary infection is relieved.

The best results by this procedure are secured in cases of bilateral paralysis complicating thyroidectomy. Cases caused by other factors

should be studied carefully particularly from the standpoint of possible endolaryngeal fibrosis or fixation of a cricoarytenoid joint.

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REFERENCES

1. King, B. T.: A New and Function Restoring Operation for Bilateral Abductor Cord Paralysis, J. A. M. A. 112:814, 1939.

2. Clerf, L. H.: The Surgical Treatment of Paralytic Stenosis of the Larynx, Plastic and Reconstruct. Surg. 2:326 (July) 1947.

PHOTO WERNER MUELLER

Sketching on board ship, 1937.

VIII

THE CRICOPHARYNGEUS MUSCLE

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Ultimus, qui restat Musculus mihi dicitur Cricopharyngaeus.
Antonius Maria Valsalva, 1717.

By christening the horizontal muscle fibers at the lower end of the pharynx the cricopharyngeus muscle, Valsalva[1] invented a new structure. His invention has not been recognized universally by succeeding anatomists, many of them like Birmingham[2] referring to the fibers as a "lower or 'oesophageal' part of (the) inferior constrictor." Variations in names may be due to usage differences in the several languages or to the traditions of a particular school of anatomy. Such variations are without fundamental anatomic significance. The cricopharyngeus muscle of Valsalva however, may have been minimized by some because of its lack of sharp definition in the anatomic specimen. A casual dissection discloses few identifying features. The fibers are horizontal and are attached to the cricoid cartilage, but very precise dissection is necessary to set the limits of the band and to isolate it from the adjacent longitudinal and oblique fibers which blend with it. In the dissecting room the selection of this bundle for a specific name has seemed unnecessary. Many have been content to talk only of an inferior constrictor muscle of the pharynx.

The endoscopic examination of the esophagus in the living, on the other hand, has left no doubt as to the validity of Valsalva's separation of the inferior part of the lowest constrictor from the rest of the muscular pharynx. In the direct examination of the esophagus the first problem to be overcome, once the use of the direct laryngoscope has been mastered, is the contraction of the cricopharyngeus muscle. The muscle shows itself by producing a sphincteric occlusion just below the pyriform sinuses of the pharynx. Unskillful handling of this problem by the inexpert has led to perforation of the posterior wall of the pharynx at this point with a succeeding, formerly often fatal, infection. Since the endoscopic examination of

the upper food passages has become well established, the term crico-
pharyngeus muscle has come into wide use.   The clinical literature of
the pharynx and the esophagus, discusses the cricopharyngeus muscle
as a separate entity.   Dr. Chevalier Jackson has emphasized the dangers
in the improper handling of this muscle in his works on endoscopy.[3, 4]
The lodging of foreign bodies at the site of the cricopharyngeal fibers,
added to the obstruction encountered here in passing an esophago-
scope, has led to the universal designation of this locus as the superior,
upper, or first constriction of the esophagus.   Jackson[3] calls it the
cricopharyngeal pinchcock.

"The cricopharyngeal pinchcock, as already mentioned, is pro-
duced by the tonic contraction of a specialized band of the orbicular
fibers of the lowermost portion of the inferior pharyngeal constric-
tor muscle, called the cricopharyngeus muscle.   As we have shown,
it is this muscle and not the cricoid cartilage alone that causes the
difficulty in the insertion of an esophagoscope.

"This constriction merits the name 'cricopharyngeal pinchcock'
because of the pinching shut of the esophageal tube by an external
muscle, whose action is similar to that of the pinchcock that pinches
shut the rubber tube on the buret.   Closure is tonic and constant;
opening is coordinate and momentary, for deglutition and emesis.
Even at complete relaxation a degree of structural narrowing exists.
Saliva in small amounts goes through the closed pinchcock but any
excess results in subconscious deglutition.   The cricopharyngeal mus-
cle relaxes at the same moment the inferior constrictor contracts, in
normal swallowing."

The custom of authors to assign the cricopharyngeus muscles to
the upper end of the esophagus or lower part of the pharynx inter-
changeably is readily understood.   The muscle is in a transitional
location and has characteristics belonging in part to the constrictor
of the pharynx and in part to the circular muscle of the esophagus.
The anatomy of the region has been much clarified by the accounts
of Birmingham[2] and Abel.[5]   The inconsistencies of some of the earlier
descriptions and the lack of agreement of some of these with our pres-
ent concepts is due no doubt to the methods of preservation in dif-
ferent periods of anatomy.   Formaldehyde was not in use as a preser-
vative until late in the 19th century.   Without the use of some method
of fixation which holds the parts rigidly in their position, it is exceed-
ingly difficult to separate minute bands of muscle.   For the best dis-
play of the cricopharyngeus muscle and its relations a solution con-
taining formaldehyde is essential.   Any other preservation gives a
specimen which is too friable, and not of sufficient rigidity for care-

Fig. 1.—Left half of an anatomic specimen of the head sectioned in the mid-line. The arrow points to the cricopharyngeus muscle.

ful work. The papers of Birmingham and Abel belong to the period in which use was being made of this better preservation to edit earlier descriptions.

The anatomy of the cricopharyngeus muscle may be summarized as follows. The fibers have their attachment to the cricoid cartilage laterally, at the junction of the annular portion with the "signet" portion of the cartilage. From these attachments the fibers from a horizontal band-like sling which encloses the mucous membranes of the lower end of the pharynx. This is at the narrowing of the pharynx into the esophagus. This band of fibers is 10 to 12 mm high. Recall that the annular or horizontal fibers of the cricopharyngeus muscle have a very short total length as they course from the cartilage back to the cartilage around the entrance to the esophagus. We have therefore an anatomic-physiologic mechanism which permits of a tremendous narrowing of the lumen by the short horizontal fibers which can contract to one-half of their length. This sphincter effect is increased since the anterior wall, the cartilage, is immobile.

I have at hand, a specimen which demonstrates the normal size and topographic relations of the cricopharyngeus muscle in contraction. The specimen here described was obtained, as are many of the best anatomic specimens, quite by accident. A large, robust, male cadaver of the sthenic habitus was embalmed with strong (20 per cent) liquor formaldehyde solution, no venous blood was withdrawn from the cadaver and the injection was continued until there was considerable distention of the tissues. After a suitable period had elapsed allowing for complete hardening of the tissues, the cadaver was frozen and cut with a power band saw.[6] The first section was made in the midsagittal plane and the remaining sections were made parallel to this. The section passing through the larynx is not in the exact midsagittal plane due either to some inaccuracy in sawing or to a slight displacement of the larynx in fixation.

To our great surprise, this section showed a prominent fold in the posterior pharyngeal wall caused by the cricopharyngeus fibers being in marked contraction. As seen from the figures (Figs. 1 and 2) the fold of the posterior mucosa almost reaches the anterior wall. The muscle bundle has a vertical height of 8 mm and an anterior posterior thickness of 12 mm. When compared with the impressions gained by examination of this muscle with the esophagoscope in the living it seems that the vertical height in this specimen is too great. Several facts should be borne in mind here. The cadaver was of a very muscular, sthenic type. No venous blood was withdrawn and a large amount of solution was used in embalming. The method of preparation may account for some of the size as the tissues do appear some-

Fig. 2.—Right side of the specimen shown in Figure 1. The arrow indicates the cricopharyngeus muscle and the mucous fold.

what distended. However the height of the "signet" of the cricoid
cartilage is 10 to 22 mm and this is certainly compatible with the
superior inferior height of the muscle.

The interest in the specimen itself was only surpassed by the
interest in what might have produced it. I have never seen such a
specimen before, neither in my own laboratory nor in any museum.
I am not familiar with any illustrations of such a specimen. I be-
lieve that the specimen is unique. There is the possibility that these
muscle fibers contracted with the onset of rigor mortis. It is a gen-
eral experience that rigor mortis is more marked in individuals of a
heavy muscular build. If this were the explanation, it seems that
these muscle fibers should be seen frequently in the contracted state.
This is not the case.

The comments of Professor Patterson[7] on the post mortem form
of the human stomach suggest circumstances that may be responsible
for the finding of this specimen of the cricopharyngeus muscle in
rigor. Patterson found the hour-glass stomach type of contraction
to be a very frequent occurrence in cadavera embalmed with for-
maldehyde. Since rigor mortis occurs first about the head and dis-
appears first there, it may be that our specimen was fixed in the early
stages of rigor mortis, or possibly the rigor was increased by the
formaldehyde. It may be that the usual cadaver has lain sufficiently
long for the rigor to have passed from the region of the pharynx.
At best, this is a guess.

To test out the hypothesis, a dog under anaesthesia was killed
and the femoral artery was isolated and perfusion of a formaldehyde
solution (20 per cent) was begun. While this fluid was flowing into
the femoral artery, the upper constriction of the esophagus was kept
under view by the use of a direct laryngoscope. As the tissues began
to blanch about the pharynx, denoting the entrance of the formalde-
hyde into them, there was a slow elevation of the vertebral wall of
the pharynx until the pharyngoesophageal junction was about one-
half pinched off by the cricopharyngeus muscle pulling forward.
How long after death this contraction could be brought about by
formaldehyde was not determined. It should be noted that our cad-
avera are at least 48 hours post mortem when received.

COMMENT

In different individuals the cricopharyngeus muscle must exist
in varying states of tonicity, indeed certain individuals appear to have
the power to inhibit the contraction altogether. Beer drinkers have
been known to throw back the head and insert a bottle of beer be-

tween the lips and to allow the gas pressure to force the entire con-
tents of the bottle down the esophagus without aid of a swallowing act.
It seems that the performer succeeds in relaxing the cricopharyngeus
muscle sufficiently to allow the entire amount to pass by unimpeded.

Those who are familiar with esophagoscopy have sometimes ex-
pressed the idea that the muscle bundle was too thick in our specimen.
It should be noted that the esophagoscopist sees but the upper surface
of the mucous membrane covering the muscle and is not in a position
to form a good idea of the vertical extent of this mass. The studies
of G.H.S. Ramsey of Rochester, N. Y., and his staff, on the swallow-
ing act, by the use of ciné-fluorography, have produced views which
closely resemble our specimen. In Dr. Ramsey's cases[8] the muscle
fold turns down as the fluid mass passes into the esophagus. I would
say that this specimen is an anatomic diagram of Dr. Ramsey's roent-
gen findings.

Elze and Beck,[9] on the basis of work of Elze,[10] thought that the
pads of pharyngeal veins made the obstruction at the lower end of the
pharynx. I repeated this work[11] and could find no basis for consid-
ering the venous pads as operating like a sphincter. The idea should
be discarded.

The picture now is quite clear. The horizontal bundle of fibers
which Valsalva named on a morphologic basis, has been established
as a sphincter of the esophagus by direct observation, by demon-
stration using ciné-fluorography, and now by an anatomic specimen
of this morphologic bundle in the contracted state.

The experimental production of a cricopharyngeus in rigor in
an anatomic specimen in the dog possibly provides a method for the
production of similar specimens in human anatomy. There remains
the physiologic investigation of nervous regulation of this muscle.
It is tempting to call the cricopharyngeus a functional sphincter,
yet all sphincters are, more or less, functional. Certainly the crico-
pharyngeus has as much anatomical entity as the obicularis oris. The
term, functional sphincter, may indicate that the anatomy of the
sphincteric fibers can not be worked out readily in the anatomic
specimens by usual means.

Accumulated clinical experience and now an anatomic specimen
justify Valsalva in marking out and naming a group of fibers, the
cricopharyngeus muscle.

SUMMARY

The cricopharyngeus muscle was marked out as an anatomic
entity in 1717 by Valsalva. Probably, because of the difficulty of

separating the muscle from the surrounding structures, this anatomic concept was not universally accepted.

The specimen here illustrated and described of this muscle band in the contracted state confirms the clinical observations and establishes the cricopharyngeus muscle as a definite anatomic physiologic structure.

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REFERENCES

1. Valsalva, Antonius M.: de Aure Humana Tractatus . . . Quibus interposita est Musculorum uvulae atque pharyngis, Utrecht 1717.

2. Birmingham, A.: A Study of the Arrangement of the Muscular Fibers at the Upper End of the Esophagus, Jour. Anat. and Phys. 33:10, 1899.

3. Jackson, Chevalier: Peroral Endoscopy and Laryngeal Surgery, Philadelphia, 1914.

4. Jackson, Chevalier and Jackson, Chevalier L.: Bronchoscopy, Esophagoscopy and Gastroscopy, Philadelphia, 1934.

5. Abel, Williamina: The Arrangement of the Longitudinal and Circular Musculature of the Upper End of the Esophagus, Jour. Anat. and Phys. 47:381, 1913.

6. Batson, O. V.: A Power Saw for the Anatomical Laboratory, Anat. Rec. 53:1:7, 1932.

7. Patterson (Prof.): The Form of the Human Stomach, Jour. Anat. and Phys. 47:356, 1913.

8. Ramsey, G. H. S.: Personal communication.

9. Elze, C., and Beck, K.: Die venösen Wundernetze der Hypopharynx, Ztschr. f. Ohrenh. 77:185, 1919.

10. Elze, C.: Die venösen Wundernetze der Pars Laryngea pharyngis, Anat. Anz. 51:205, 1918.

11. Batson, O. V.: Veins of the Pharynx, Arch. Otolaryng. 36:212-219, 1942.

THE PROBLEM OF POSTNASAL DRIP

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Browsing recently through a number of the Journal of the American Medical Association, weeks after the time when I should have read it, my eyes lighted on the page entitled "Queries and Minor Notes," and so on the following letter:

"To the Editor:

One of the commonest ailments of mankind is the postnasal drip, the profusion of mucus that accumulates exactly at the point of the gag reflex in the nasopharynx. Day after day the victims of an "inconsequential" (or so patients are told by the doctor) affliction go through hours of discomfort from nausea and vomiting until the mucus can be dislodged from the gag reflex area and expectorated. Is there any successful treatment for this condition? I have done innumerable patch tests, used all the antihistamines, forbidden smoking and the use of condiments, all to no avail. The picture of the otherwise healthy patient who every morning and often several times a day must cough, gag, and vomit because of a localized accretion of mucus is a sad commentary on modern medical practice. We make much of our progress against cancer and other diseases but the thousands of persons condemned to their daily vomiting spell find no surcease because theirs is not a killing illness. Is there not someone in our profession who knows the answer?"

—signed, M.D., New Jersey.

This plaintive voice as of one crying in the wilderness will I am sure strike a sympathetic chord in the heart of any physician who day after day is consulted by patients whose often sole complaint is that they have "a dropping in the back of the throat," or what in days of yore was customarily designated as "catarrh." It might interest the writer of this appealing letter to know that his problem is no new one, or the outcome of our present mode of living. Even Galen was confronted by patients with "mucus, which if especially viscid, is hawked out through the mouth."

I have no statistics but from my own experience I would hazard a guess that ten to fifteen per cent of any laryngologist's practice is

made up of individuals who voice this symptom. Before returning
to utilize this anonymous appeal as a text for my sermon, I should
like first if possible somewhat to clarify the meaning of the term
"postnasal drip," and so avoid an endless and labyrinthine discussion
of nasal discharge in general. What then may properly be designated
by this term of postnasal drip? Let us look at it first of all from the
patient's point of view, to which, I regret to say, I can bring some
personal observations. The chief complaint is a sensation, localized
roughly to the posterosuperior surface of the soft palate, of some-
thing which is not normally present and which therefore generates
the desire to be rid of it. Vaguely it is a sense of fullness, not quite
so much as to constitute the otherwise frequent description of a lump,
but of something which should be readily dislodged by swallowing.
It being immediately evident that such swallowing is quite useless in
eliminating the sensation, there is next evoked the peculiar and more
vigorous pharyngeal reaction which has come to bear the odd term
of "hawking" and which consists in an attempt, by fluttering the soft
palate against the posterior pharyngeal wall, to dislodge whatever
seems to be lodged in this sequestered area. Such hawking is usually
productive of momentary relief because of a feeling that mechan-
ically something positive has been accomplished but the very next
swallowing movement is more than likely to find the same disagree-
able sense of an intruding sensation still present. Indeed, constant
efforts thus to dislodge a presumed collection of viscid secretions may
generate an edema of the soft palate which in itself provokes a similar
sensation even when no secretions are present. In such a situation
the application of a weak anesthetic to the posterior surface of the
soft palate will promptly eliminate all discomfort for a short period.

Repetitious hawking tends soon to generate a vicious cycle and
hence in many individuals with a sensitive nasopharynx, to lead by
a sort of tripping mechanism to the stimulation of gagging and the
latter, if sufficiently active, to the vomiting about which our com-
plainant is so exercised that he mentions it no less than three times in
his short communication. It is largely a matter of degree. For a
majority of patients it is simply a form of annoyance with which
they can if necessary put up but for which they seek relief in the
hope that some simple measure will eradicate it. For others it is a
source of real distress, seriously interfering with their enjoyment of
eating and with their sleep and even at times plaguing them to dis-
traction. I am doubtful, however, if by and large it really induces
in any except a few exceptional instances the "daily vomiting" to
which reference is made in the letter. The real crux of the symptom,
somewhat vaguely described because it is only thus vaguely that the
patient usually can describe it, is, if Webster will sanction a fabricated

word, the "undislodgeability," or better still, the non-fluidity of whatever it is which seems incarcerated in the area behind and above the soft palate. As the patient puts it, "I can't seem to get rid of it."

Intermittantly he will be aware of something suddenly letting go as it were, in all probability a mass of mucus, and of its sliding down into the lower pharynx whence it can be easily swallowed or expectorated and following which he senses an immediate relief of his discomfort. This spontaneous dislodgement otherwise voluntarily unachievable, is doubtless what has given rise to the "drip" component in our terminology of "postnasal drip." Paradoxically if there were only more dripping, more spontaneous detachment of the offending mass of mucus, the patient would be far more comfortable. Again let me stress at this point that his chief complaint arises from his inability to effect such detachment at will, which in turn leads to more and more vigorous, albeit, ineffectual efforts to accomplish this.

Basically, from a patient's point of view, that is the very hub of his complaint. He does not mention nasal obstructions or a sore throat. Least of all does he complain of any anterior nasal discharge. His handkerchief is regarded only as a receptacle into which to hawk down something from his mouth. The significance of the absence of such other complaints I shall return to in the consideration of what is primarily *not* postnasal drip.

How now does the matter look to the examining laryngologist, who having listened to this story, often I fear with an already well established feeling of frustration, proceeds to the examination of the patient? By and large, and with acknowledged exceptions, I think it is fair to say that if such an examination were conducted before taking the patient's history, the physician would have but a vague idea of the nature of his complaint, or indeed that he had any complaint at all referable to his nose and throat. The posterior pharyngeal wall appears covered only with a normally thin film of clean mucus. There is a free airway on each side of the septum, the turbinates are of normal size, their mucosal surface is normal in color and apparently covered with the same clear mucous film seen in the nasopharynx. Transillumination of the sinuses is negative. While further and much more detailed diagnostic procedures which I shall describe later, must be routinely carried out, the laryngologist is already in far more of a diagnostic and therapeutic dilemma than if the patient were complaining of unilateral purulent discharge, bilatteral nasal obstruction, or frontal headache, any one of which would be more likely to be accompanied by definitely objective findings on examination than is true of postnasal drip.

Therefore, let me now exclude certain conditions, which, though sometimes listed as causing postnasal drip, do not fall within at least my definition of this complaint. The first of these is true sinusitis, productive often of nasal discharge, usually purulent in character and frequently seen running down the posterior pharyngeal wall. Oddly enough, in the face of such postnasal discharge, to be distinguished I think from postnasal "drip," there is repeatedly no complaint of its presence by the patient. He complains that he blows out purulent discharge from one side of his nose and exhibits his one or more handkerchiefs to prove it. He has headache, a unilateral sense of discomfort over the affected sinus or nasal obstruction of which he is far more likely to be aware than of the discharge in his nasopharynx.

The second great clinical entity to which is so often given a major role in the causation of postnasal drip is allergy. Conceived of today as a possible cause of almost any nasal abnormality, its fundamental manifestation of profuse watery nasal secretion, its bilateral turbinal enlargement and resultant nasal obstruction and as we shall see later, the typically high percentage of eosinophiles in the resultant discharge, are rarely associated with the picture which is continually seen in patients complaining of postnasal drip.

A third condition, admittedly productive for obvious reasons of nasopharyngeal discomfort, is atrophic rhinitis, often listed quite rightly as a cause of postnasal discharge, but having no other similarity to the condition with which I am here concerned.

In complete disagreement with this point of view is a forceful Irishman from Cork who believes, "that all postnasal discharge is due either to sinusitis, atrophic rhinitis or hypertrophic rhinitis. Essential nasopharyngeal catarrh (presumably his term for postnasal drip) is of such rarity as hardly to merit practical consideration. In its chronic manifestations x-rays will always show ethmoiditis."

If, however, one is willing to accept my somewhat limited definition of postnasal drip, if one will agree to the essential features of its symptomatology, and if one excludes what may be termed rival conditions, basically of a different and more easily determined etiology, I should like now to inquire somewhat into the nasal physiology which underlies these complaints, which, I fear with some justification, constitute what our correspondent characterizes as a sad commentary on modern medical practice and possibly on rhinolaryngology in particular.

PHYSIOLOGY

It has long been known that the nose functions as an air-conditioning organ, in the process of which there is secreted by the glands of the nasal mucous membrane covering the turbinates and meatuses and lining the sinuses, a mucus fluid consisting of 3 per cent mucin and 97 per cent water, mucin itself being a complex combination of protein and glucose. This mucous in the form of a thin sheet or blanket is constantly being swept over the nasal surfaces by ciliary action and passes backward into the nasopharynx where it is unconsciously swallowed. It takes only a small increase in the mucinous content of nasal mucus materially to slow down this ciliary action, and thus to impede its progress toward discharge into the nasopharynx. This mucus blanket, according to Hilding, is renewed over the posterior two-thirds of the nose every ten minutes and over the anterior third approximately every hour, a degree of production which amounts to from one to two quarts of mucus secreted per day. Depending on the environmental humidity, a portion of the fluid is being constantly evaporated, thereby to a varying degree diminishing the amount which reaches the nasopharynx. The secretory process is entirely under the control of the autonomic nervous system and any one of a wide range of factors which disturb autonomic function will affect the amount of mucus secreted and its physical and chemical constituents. It is such disturbances which lead to the symptom complex which we are now discussing. So long as a normal amount of mucus, secreted at a normal rate and of a normal viscosity and chemical composition can pass unimpeded by structural deviations, an individual is essentially unconscious of this whole physiological and automatic process. Derangement of one or more of these fundamentals results in what our correspondent has described as "one of the commonest ailments of mankind."

Unlike the blood, the lymph, or spiral fluid, nasal mucus is in a state of normal fluctuation because of its subjection to constantly changing conditions under which it is produced and external factors by which it is influenced. As Parkinson says, "Nasal secretion is wholly variable both in composition and in physical values. It arises as a secretion, excretion and exfoliation from the entire surface of the nasal tract. It consists of mucoid fluid with inclusion of epithelial and white blood cells and their toxins and foreign substances of inhaled origin. It is subjected to air currents of varying temperature and humidity and carbon dioxide content, and is modified by autolysis, decomposition and fermentation and by the products of growth and death of pathogenic and saprophytic bacteria. Being lifeless material it has no means of maintaining the standards of sur-

rounding tissues and its composition and characteristics at any given time are unpredictable."

Indeed the wonder is that this complicated mechanism, whose normal function is subjected to such variables and unpredictables is ever in a state of normality. Certainly no mechanical air-conditioning device yet invented could meet such high standards of performance and the capacity of the nose automatically to adapt itself at one moment to the climate of Hawaii and at the next to the ceaseless fluctuations of a rugged New England winter is reason indeed for amazement. The same basic mechanism must serve the laundry worker and the refrigerator attendant, maintaining a proper balance in the moist environment of the florist and the dessicated atmosphere surrounding the office worker. It is hardly to be wondered at that the machine occasionally shows signs of running a bit less smoothly than usual. As Lillie has said, "The understanding of the mechanism of the function of the nasal mucous membrane, and its reactions to various environmental conditions is fundamental in the practice of rhinology." Perhaps to the many items of a rhinologist's training we should add a course in air-conditioning.

Since the production and physical characteristics of nasal mucus are under control of the autonomic nervous system, it is not surprising to find individuals who are more susceptible than others to situations tending to upset this control. Some people sweat more easily, blush more quickly, palpitate more readily than others. My own pulse rises 20 points on approaching the first golf tee. Vasomotor instability finds a common and ready expression in nasal dysfunction as will appear later. Such instability, congenital or acquired, may render many individuals constitutionally unable to adjust readily to situations to which others, more stable, are completely indifferent.

The writer of our introductory letter has referred to his patients with postnasal drip as "victims of an inconsequential affliction." By this I assume that he means that they have been led to infer that their physician considers it inconsequential and hence is not duly sympathetic toward their complaint. I fear that at times, I too, after reading in the morning paper of some father returning from work to find his home and three children burned to the ground, have regarded the complaint of postnasal drip as relatively trivial. Nevertheless, it is a bona fide symptom for which people are seeking relief and for which they are entitled to the best service we can give them. To this complaint, Arthur Proetz, himself a master in the field of nasal physiology, has given the colorful designation "The Current American Nightmare." This phrase would at once suggest that such afflicted individuals are terrified by their complaint, much as are people who

fear that their symptoms may stem from cancer and suggestions are frequently made that one has only to reassure them as to the fundamentally innocuous nature of postnasal drip to dispel such fear and thereby automatically to effect a cure. This in general has not been my experience. It is true that as a result of misinformation of friends and the lay press, many persons suspect their symptoms to be indicative of sinus disease and that reassurance on this score and as to the harmlessness of swallowing normal mucus will make them happier, but they will usually continue to complain and request relief from a symptom, which although not serious, is most annoying and which occasions them much discomfort.

Excessive amounts of nasal secretion are usually the transitory response to some external environmental irritants such as smoke fumes, chemical vapors and the like. Nasal discharge is likely to operate obstructively only if present in a nose of which the passages are encroached upon by some anatomical obstruction such as is produced by a deviated septum or an enlarged turbinate. It is the increased viscosity of otherwise normal nasal mucus which is by far the most common cause of the complaint of postnasal drip, since it operates to prevent the patient from removing the mucus from an area where its lodgement creates a distressing sense of discomfort.

CYTOLOGY

The microscopic examination of the cellular constituents of postnasal mucus affords little of conclusive value in our efforts to solve our problem. In a study of 100 consecutive clinic patients, reported by Moos in California, where we in the East are lead to believe that all is perpetually well with the nose, 50 per cent complained of postnasal discharge. Microscopic examination of this discharge in all these 100 patients showed one-third with no cells of any sort. Of the 50 patients complaining of discharge, 86 per cent showed polymorphonuclear cells and 22 per cent eosinophiles. In the 50 patients without postnasal discharge, however, 40 per cent showed polymorphonuclear cells and 14 per cent eosinophiles, indicating that even normal mucus frequently contains such cellular constituents. There was, in general, about an equal cellular content in patients showing no signs of nasal pathology as there was in those with demonstrable disease.

I should like now to examine some of the causes commonly ascribed to this specialized form of nasal discharge. They may, I think, helpfully be divided into factors which affect the patient either environmentally or which stem from causes affecting him as a whole. They may conveniently be called extrinsic factors and be listed as follows:

1. Atmospheric
2. Stimulants and irritants
3. Emotional
4. Endocrine and metabolic
5. Dietary

Let us consider these individually:

1. *Atmospheric.* Postnasal drip is, in my experience, far more common in the winter months than in the summer, and at least in New England, is related to our over-heated and under-humidified dwellings and offices. Many people for months are subjected to an atmosphere of under 30 per cent humidity, and the nasal mucosa is constantly called upon, often unsuccessfully, to supply the necessary increased moisture to overcome this situation. In the face of excessive dryness of the air, the mucinous content of the nasal secretions is relatively increased with a resultant increased viscosity that produces the commonest basis for our correspondent's complaints. The vogue for fresh air (and so relatively dry air) during sleep, when all physiological activity is slowed down throws an additional burden on the nose and explains the nasopharyngeal accumulation of mucus, of which on arising, the patient finds himself obliged to rid himself by the unpleasant ritual of repetitious hawking and wretching. Dwellings and offices may some day be universally air-conditioned, with benefit alike to occupants and furniture.

2. *Stimulants and irritants.* Smoke, dust, fumes and occupational irritants are common causes of an increase in nasal secretions, but they do not, in general, lead to the viscosity and difficulty of dislodgement which here concern us. As for tobacco and alcohol, always high on a list of etiological factors, they serve, I believe, chiefly as handy proscriptions which the physician, probably quite beneficially, can impose on his patient, but without any great liklihood that they are directly responsible for his nasal symptoms.

3. *Emotional.* We live today in a world of emotional and nervous tension, and a psychosomatic attitude toward symptoms once considered unequivocably organic. It has been clearly proved that disturbances of psychic and emotional origin can affect the nose and its secretory activity just as much as the gastrointestional tract or circulatory system. Wolff's findings on this subject occupy a full-sized book and so serve to complicate the rhinologists problem as to how far he is to go in the semi-psychiatric investigation of his patient. Unquestionably, some postnasal drip is of this nature, though few patients will welcome a diagnosis which seems to them to stamp them as neurotic.

4. *Endocrine and metabolic.* The complications of this avenue of approach to our problem are obvious. The influence of the ductless glands on our whole sympatheic nervous system is so great that to associate nasal hypersecretion and changes in the constituents of nasal mucus with increased or diminished activity of the thyroid gland, the adrenal or the pituitary, is a very natural and often justifiable temptation however difficult of scientific proof. Changes in nasal secretion during pregnancy or the menopause have often been observed, and frequently offer a welcome explanation for the patient's complaint. Today an otolaryngologist's training must needs apparently be broad enough to include even a little gynecology.

5. *Dietary.* Some fifteen years ago there swept over certain parts of the country, largely by mail, a lively interest in the influence of dietary factors on the causations of noninfectious nasal discharge. Patients were catalogued as alkaline or ash ingestors and forthwith presented with a list of foods of opposing acidity or alkalinity. In general, if one suffered from "catarrh," if I may be pardoned the use of this outmoded term, he shuffled his intake from whichever side of the neutral line it originally lay on. Hydrochloric acid and soda bicarbonate were revived after years of therapeutic oblivion, citrus fruits were exchanged for meat, and bananas replaced bacon. There was talk of the biochemical approach to rhinology, despite the unsympathetic and even critical attitude of the biochemist and physiologist. Nevertheless, there were numerous and probably quite unbiased reports of clinical success in the applications of this therapeutic approach which offered an appealing and simple method of achieving results. Unfortunately it was quite incapable of anything approximating controlled laboratory accuracy and it stood or fell largely on patients' statements, on clinical observations and on the rhinologist's personal enthusiasm, buttressed, I fear at times, by some degree of wishful thinking. Nevertheless, there was and is today a large modicum of truth in the belief that the amount and quality of nasal secretions can be materially influenced by dietary modifications, quite apart from ancillary systemic benefits from such loss of weight as might result from rigid exclusion of all sugar and carbohydrates from the diet.

It is surely not difficult to appreciate the diagnostic dilemma of the rhinologist who is confronted on a cold New England afternoon by a woman in the menopause, unhappily married, with a deviated septum and inordinately fond of chocolates, who is seeking relief for "a dropping in the back of the throat."

Is she to be told to put pans of water on the radiator, to take hormone injections, to leave her husband, or to give up candy? I

submit that this poses no easy problem if one is to give due considera-
tion to the possible etiological factors which may lie at the root of
the problem. Admittedly, in a majority of instances, such postnasal
dripping, or, more accurately, failure to drip, is merely annoying and
does not from a patient's point of view warrant any serious disturb-
ance of an otherwise comfortable manner of living. Hence his hope
of a prescription for some spray, pill or drops rather than instructions
to stop smoking or to eliminate all starch from the diet, much less to
move to another part of the country or to break up his home. Yet
only by time-consuming inquiry into a patient's type of work, his
living and eating habits, his modes, if any, of exercise, his mental
strains and emotional stresses, can one properly evaluate the possible
causes of this so-called "inconsequestial" affliction.

Let us turn now to what one may call the local or purely organic
conditions which occupy every list of supposed causes of postnasal
drip and which, as submitted in the Journal's answer to our plaintive
correspondent, may well have left him even more confused and be-
wildered than before writing his letter.

1. Sinus Infections and Allergy.

2. Structural Abnormalities.

      A. Deviation of the Septum.
      B. Turbinate Hypertrophy.
      C. Lymphoid Hyperplasias.
      D. Cysts, Scar Tissue, Mucosal Metaplasia.

SINUS INFECTIONS AND ALLERGY

I have already made it clear that I do not believe that ordinarily
the purulent discharge seen in the nasopharynx and emanating from
an inflamed nasal sinus, nor the profuse watery discharge which cus-
tomarily characterizes nasal allergy are the source of what most pa-
tients describe as a dropping in the back of the throat. These con-
ditions are productive of nasal discharge but I must to this extent
draw a sharp distinction between discharge and drip, similar though
they may at first sight appear. In this respect I cannot agree with
Van Alyea, his eminence in the field of rhinology notwithstanding,
when he says that few patients complain solely of postnasal drip, the
commonly presenting symptoms being those of sinusitis, sore throat,
hoarseness and cough, findings which are completely the reverse of
my own. If, as claimed by the aforesaid gentleman from Cork, post-
nasal drip is one hundred per cent the result of ethmoiditis, then this
diagnosis must be made in the absence of any of the accepted criteria

of this condition, and we must designate as ethmoiditis any slight increase in the normal out-put of mucus from these sinuses.

STRUCTURAL ABNORMALITIES

A. *Septal Deviations.* Mechanical obstruction to nasal air currents, septal spurs and ridges, particularly if impinging on turbinal surfaces are repeatedly listed as causative factors in postnasal drip, on the theory that the resultant disturbance of air currents induces a metaplasia of the mucosa from a ciliated columnar to a squamous epithelium with production of a dryer and thicker type of mucus. Such septal deviations may well be so relatively minor as to provoke no complaint of nasal obstruction, and so pose a problem as to when they are worthy of operative correction. Since a perfectly perpendicular septal partition is almost an anatomical curiosity it is no easy matter to decide when minor departures from the straight and narrow are responsible for a complaint which, as I have already indicated, may well be due to far different causes, seemingly much less related to the nose. Decision here calls for keen judgment and a stout conscience and allows small room for wishful thinking.

B. *Turbinal Hypertrophy.* Turbinal hypertrophy if primarily edematous or hypertrophic, is far more likely to provoke a complaint of nasal obstruction than of postnasal drip. Doubtless, such hypertrophies are accompanied by increased glandular secretion, and by encroachment on normal air ways, interfere with free passage of secretions into the nasopharynx with resultant stasis and so increased viscosity. Edematous enlargement of the posterior tip of the inferior turbinate tips has been invoked by one West Coast rhinologist as the really essential factor in postnasal discharge and as amenable to the intriguingly simple procedure of repetitious cauterization with carbolic acid. If the problem were indeed so simple, it would be unnecessary to discuss other more complicated approaches to it. It has however, frequently surprised me on routine nasopharyngoscopic examination to note a pale polypoid enlargement of the posterior turbinal tip in complete absence of complaints either of obstruction or postnasal discharge, even when strands of mucus could be seen stretching from the turbinate to the posterior pharyngeal wall. The application of any of the common procedures designed to reduce the size of the inferior turbinate (chemical cauterizations, diathermic coagulation, or surgical removal) may achieve a patent airway when this is the prime objective but as a treatment for postnasal drip, they are by no means assured of success.

C. *Lymphoid Hyperplasia.* Postnasal drip is not infrequently seen in the presence of hypertrophied and isolated islands of lymphoid

tissue on the posterior pharyngeal wall, of enlarged lateral bands of lymphoid tissue back of the posterior pillars and of a varied sized mass of adenoid tissue. To assume that these lymphoid masses are themselves responsible for the discharge is to my mind no more logical than to assume them to be the result of the irritation produced by constant efforts to dislodge the discharge. The presence of such tissue may well give rise to sensations closely resembling those of postnasal drip, in which case removal of one or more of these masses of lymphoid tissue may rid the patient of his symptoms. Hence the questions of which is cart and which is horse is not easy to answer.

D. Cysts, Scar Tissue, Mucosal Metaplasia. The presence in the nasopharynx of postoperative scar tissue, of medial cysts or a Thornwald's bursa and chronic irritation of the mucosa of the pharyngeal vault may be associated with mucoid discharge and so at times be productive of the complaint of postnasal drip.

In any case, the presence or absence of what I have chosen to call local or organic conditions which may be associated with postnasal drip can only be determined by a thorough examination of the nasopharynx by the following routine measures.

1. The nasopharyngoscope, the use and value of which are well known to all rhinologists.

2. The Yankauer nasopharyngeal speculum, which exposes a view of the posterior pharyngeal wall to direct vision and facilitates any manipulations which may be necessary in connection with it.

3. The pharyngeal mirror and the soft palate retractor, without which the mirror will more often than not prove useless because of the close proximity of the soft palate to the posterior pharyngeal wall, and the highly sensitive gag reflex present in most patients complaining of postnasal drip. Both the Yankauer speculum and the self-retaining palate retractor require thorough cocainization of the nasopharynx, which may easily consume a half hour or more. In no other way, however, can an adequate examination be made of this region in an effort to eliminate local causes of postnasal drip. A forerunner of such a palate retractor advocated 50 years ago combined a mechanism for holding open the jaws, depressing the tongue and withdrawing the soft palate in one instrument, which would not, in my opinion, be tolerated by one in fifty patients.

THERAPY

It may comfort our perplexed correspondent to learn that his problem is no new one and that seventy years ago in a book entitled "Postnasal Catarrh," Edward Woakes was searching for the thera-

peutic answer. He was evidently a firm believer in good general hygiene, since obviously long before the days of modern plumbing, he advocated that the patient each morning stand in a foot-bowl of warm water, sponge all over with a cold solution of laundry soap, and dry with a rough towel while still standing in the warm water. He recommended clothing of some wool content throughout the year, the proportion to be varied with cotton according to the season, except that underclothing should be woolen for four-fifths of the year and the socks perpetually so. The bedroom should be warmed with a fire and the feet with a hot water bottle or woolen socks. Anticipating by fifty years the dietary approach, Woakes advised a limited ingestion of sugar, especially if refined, a freer use of fresh fruits and vegetables and a greater intake of water. He stressed the value of outdoor exercise, certainly much more neglected today than seventy years ago, and praised the riding of tricycles as requiring both skill and nerve.

Even at the turn of the century we find so distinguished a rhinologist as Holbrook Curtis appreciative of good general health as a fundamental in any complaint of disordered nasal secretion. For his patients thus afflicted he prescribed 28 Sandow gymnastic exercises morning and evening and a walk of ten blocks, presumably New York blocks, after breakfast and lunch, increasing a block a day to an unspecified limit. Not content with this vigorous regime, he advocated a rectal sitz-douche for the colon, night and morning, with four quarts of water! An apparatus for this purpose, once manufactured in Maine, bore the colorful designation of "The Cascade Enema Bag."

It is more than likely that such a health program would prove highly beneficial to our correspondent's victims of an "inconsequential affliction," or to most of us today who dash about in automobiles and make no pretense of regular exercise, but one wonders if modern life affords quite enough time for carrying it out. The wide range of local therapy which the modern rhinologist utilizes in the office treatment of postnasal drip serves only to emphasize the lack of any remedy which can be said to be of really dependable value.

And so in replying to the anonymous writer of the letter, we will, I fear, have to answer somewhat as follows:

"Dear Doctor:

We sympathize with your dilemma. We too have wondered what to do about postnasal drip and why such an "inconsequential affliction" should pose such a diagnostic and therapeutic problem. To it we have come to believe there is probably no simple and specific answer, sur-

gical or medical.   To it through the years, from the days of the
pioneer rhinologists, men have applied themselves with unstinted zeal,
and have long since tried and often discarded many procedures and
ideas which have since been revived as newly discovered and original.
Ultimate solution of your problem will probably come if at all through
the collaboration of several groups of workers in allied fields.   To para-
phrase a recent medical best seller,[1] we must today, as did King
Nebuchadnezzer of old, summon the magicians, the astrologers, the
sorcerers and the Chaldeans from the immunological and biochemical
laboratories, from the diet kitchens and from the departments of
physiology and psychosomatic medicine, in order that they may all
perform their mysteries and work their incantations.   Even with
their help, you will, I suspect, continue to see patients plagued by
postnasal drip in whom you will still be able only to guess at its etiol-
ogy, and to hazard a successful treatment.

77 MASSACHUSETTS AVE.

REFERENCE

1. Irving, F. C.: Safe Deliverance, Houghton, Mifflin, 1942.

X

LABYRINTHINE SURGERY IN THE TREATMENT OF
MÉNIÈRE'S DISEASE

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AND

K. H. SIEDENTOP, M.D.

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This paper presents the experience at the University of Chicago Clinics with different types of labyrinthine surgery for the prevention of dizzy spells in cases of Ménière's disease. The report covers all cases operated upon from 1940 to 1954 in the Ear, Nose and Throat Department. The main purpose is to review the results of the various operations and to describe the surgical procedure which has been selected on the basis of this experience. Twenty-five cases were operated upon, five of which required a second operation while a sixth case was reoperated elsewhere. It is estimated that less than ten per cent of all cases of Ménière's disease seen in that period of time were treated by surgery. In a report made in 1948[1] the labyrinthine surgery which had been done at this institution for Ménière's disease was reviewed and results of animal experiments presented. A number of clinical cases were then described which for completeness are included in the tables in this report.

The indication for surgery in all cases was the failure of medical therapy to prevent dizzy spells which were of sufficient severity and frequency to interfere with the patient's ability to carry on his occupation.

In all cases there were auditory disturbances of the type which is characteristic of Ménière's disease.

A threshold loss of hearing either for low tones or in more advanced cases for all tones was present in the affected ear.

Varying degrees of fluctuation in thresholds for pure tones were shown in most cases. Recruitment of loudness as demonstrated by the loudness balance technique, and a relatively low maximum discrimination score for phonetically balanced words were demonstrated in the more recent cases.

Displacusis was noted in some cases and hypersensitivity to loud sounds was complained of by a high percentage.

REVIEW OF RECENT REPORTS ON SURGICAL PROCEDURES
FOR RELIEF OF MÉNIÈRE'S DISEASE

R. L. Flett[2] gave a follow-up of 60 cases of Ménière's disease in which the Portmann procedure was done. Results were considered good in 35, improvement in 12 and poor in 13.

W. S. Adams[3] reported no improvement in one case after Portmann's procedure.

Barber and Ireland[4] gave a follow-up of 103 cases operated upon by McKenzie by differential section of the eighth nerve. Attacks of vertigo were completely relieved in 83 per cent, the tinnitus was improved in 41.5 per cent. Fifteen patients out of this group had been operated upon between 10 and 20 years earlier. Four of these retained useful postoperative hearing for an average of seven and one-half to eight years.

Green and Douglas[5] reviewed the cases operated upon by section of the eighth nerve (131 cases) and of the vestibular division (277 cases) by W. E. Dandy. Of the latter group over 90 per cent were free from vertigo; hearing was improved in 9.5 per cent, unchanged in 28.2 per cent, absent in 13.8 per cent and worse in 48.5 per cent. A transient facial paralysis occurred in 5.8 per cent and a permanent facial paralysis in 1.7 per cent.

Lathrop[6] reported 25 cases of total section of the eighth nerve and 66 cases of partial section done by the neurosurgical service at the Lahey Clinic. Of the latter 66 cases 46 were relieved of vertigo, five were not improved and 13 could not be traced. Tinnitus was not benefited. Of the 25 cases with total section of the nerve, vertigo was relieved in sixteen, unchanged in four while the others could not be traced. Lathrop also reported on 20 cases operated upon by Day's method of electrocoagulation and on five cases operated upon by Cawthorne's method. Vertigo was relieved, and complete deafness resulted in all. Tinnitus was improved in the latter group but persisted in five of the former group.

Castellano[7] gives a report of 300 cases. He sectioned the eighth nerve completely in 27 cases. All became deaf, and free of vertigo. In 226 cases section of the vestibular portion was done. All patients became free of vertigo except those with bilateral disease; forty-six became totally deaf and in all cases the tinnitus was little changed. There was no operative mortality but a temporary facial palsy oc-

curred in 14 cases and permanent palsy in two. The follow-up period extended between a few months and 19 years.

Day[8] reported his experiences on 94 cases on which he had carried out a destructive operation on the labyrinth. He had used his coagulation technique on the earlier cases, but on later cases had used a dental broach to destroy vestibular structures followed by coagulation with a fine needle electrode. He recommended actual removal of a part of the labyrinth as the surest way of destroying function and advised against attempting to preserve hearing. He believed that persistence of the low-pitched tinnitus was due to inadequate destruction of the cochlea.

Altman[9] reported 15 cases treated by coagulation of the membranous horizontal canal. Vertigo was relieved in all, hearing completely lost in eleven and decreased in four and tinnitus was unchanged in five but absent or improved in ten.

Passe and Seymour[10] reported on stellate ganglionectomy on the same side in unilateral Ménière's disease. They reported on 12 cases with relief of vertigo in all.

Passe[11] reported on 88 cases after dorsal sympathectomy. Eighty-two per cent were reported free from attacks of vertigo.

Frenckner[12] reported results on 30 cases after fenestration, plugging the vestibule with chips of cartilage, section of the chorda tympani nerve and tympanosympathectomy. Vertigo disappeared in all cases.

Lempert[13] reported on removal of the stapes and round window membrane in 15 cases by the transtympanal approach. All were relieved of vertigo while tinnitus persisted in three.

Rosen[14] reported on section of the chorda tympani and Jacobson's nerves by the transtympanal approach in 14 cases. Hearing was not changed. Vertigo was reported as relieved in 11 cases. Tinnitus was relieved in one case.

Cawthorne[15] reported on 159 cases treated surgically. Since 1938 he has removed the incus, made a fenestra and removed with a small forceps a piece of membranous canal and its ampulla. In 152 cases cochlear and vestibular function were entirely abolished. There were no records on seven. One-hundred and forty cases were able to resume work while twelve remained unimproved and unable to work.

Goodyear[16] reported relief from vertigo by making a fistula in the horizontal canal and puncturing the membranous labyrinth (utricle) with a barbed dental broach.

TABLE I.—PORTMANN OPERATION.

CASES	HEARING	VERTIGO	TINNITUS	FOLLOW-UP
P. H.	Unchanged	Recurrence	Unchanged	48 months
W. I.	Impaired	Relieved	Improved	42 Months

FENESTRATION AND DECOMPRESSION OF PERILYMPHATIC SPACE

CASE	HEARING	VERTIGO	TINNITUS	FOLLOW-UP
B. J.	Impaired	Recurrence	Unchanged	7 months

FENESTRATION OF MID-PORTION OF HORIZONTAL AND SUPERIOR
CANALS WITH DESTRUCTION OF MEMBRANOUS CANALS

CASE	HEARING	VERTIGO	TINNITUS	FOLLOW-UP
P. H.	Destroyed	Relieved	Improved	26 months

FENESTRATION OF HORIZONTAL CANAL AND INTRAVESTIBULAR
NEEDLING OF SUPERIOR DIVISION OF VESTIBULAR
NERVE WITH BARBED DENTAL BROACH

CASES	HEARING	VERTIGO	TINNITUS	FOLLOW-UP
R. M.	Destroyed	Relieved	Improved	60 months
R. G.	Destroyed	Relieved	Relieved	114 months
B. E.	Destroyed	Recurred	Unchanged	14 months
H. E.	Destroyed	Relieved	Improved	108 months

DESCRIPTION OF SURGICAL PROCEDURES

Several factors prompted a renewed search about fifteen years ago for some type of surgery on the labyrinth which would prevent dizzy spells in Ménière's disease without sacrificing hearing. Chief among these were the success of the fenestration procedure for otosclerosis, the reassurance against infections provided by sulfonamides, and the large percentage of unsatisfactory results from medical therapy.

TABLE II.—DAY OPERATION.

CASES	HEARING	VERTIGO	TINNITUS	FOLLOW-UP
G. C.	Destroyed	Recurrence	Unchanged	24 months
C. J.	Destroyed	Relieved	Improved	48 months
C. K.	Destroyed	Relieved	Improved	48 months
W. C.	Destroyed	Relieved	Improved	48 months
A. F.	Destroyed	Relieved	Unchanged	54 months
K. E.	Destroyed	Relieved	Unchanged	120 months
W. G.	Destroyed	Relieved	Unchanged	2 months
N. M.	Destroyed	Recurrence	Improved	72 months
B. J.	Destroyed	Relieved	Improved	12 months
	9 destroyed	7 relieved 2 recurrence	5 improved 4 unchanged	

The experiments on monkeys reported in 1948[1] indicated that the vestibular mechanism could probably be destroyed without causing histologically demonstrable damage to the cochlear apparatus. The earlier operations reported in 1948 represented efforts to preserve hearing and at the same time prevent attacks of dizziness. The later cases were operated upon after it was recognized that it was not possible to destroy the activity of the vestibular sense organs while consistently preserving the hearing and furthermore that tinnitus, a major complaint, could more probably be relieved if the destruction of the sense organs was complete.

Seven of the eight cases summarized in Table 1 were included in the 1948 report.[1] The eighth case was the second in which the Portmann operation was done. These operative procedures were given

LINDSAY-SIEDENTOP

TABLE III.—CAWTHORNE OPERATION.

CASES	HEARING	VERTIGO	TINNITUS	FOLLOW-UP
E. J.	Destroyed	Relieved	Relieved	14 months
P. J.	Destroyed	Relieved	Improved	9 months
D. H.	Destroyed	Relieved	Relieved	1 month
C. G.	Destroyed	Relieved	Improved	24 months
H. S.	Destroyed	Relieved	Relieved	60 months
G. X.	Destroyed	Relieved	Unchanged	9 months
S. M.	Destroyed	Relieved	Unchanged	36 months
P. R.	Destroyed	Relieved	Unchanged	8.5 months
B. E.	Destroyed	Relieved	Improved	2 months
T. R.	Destroyed	Recurrence	Unchanged	15 months
10	10 destroyed	9 relieved 1 recurrence	3 relieved 3 improved 4 unchanged	

TABLE IV.
EXENTERATION OF HORIZONTAL AND SUPERIOR CANAL AMPULLAS AND CONTENTS OF VESTIBULE.

CASES	HEARING	VERTIGO	TINNITUS	FOLLOW-UP
T. R.	Destroyed	Relieved	Improved	15 months
M. J.	Destroyed	Relieved	Improved	6 months
A. V.	Destroyed	Relieved	Improved	18 months
G. C.	Destroyed	Relieved	Improved	24 months
4	4 destroyed	4 relieved	4 improved	

up because of the failure to consistently prevent dizzy spells and at the same time preserve hearing.

Eight of the nine cases included in Table 2 operated upon by Day's method were also mentioned in the 1948 report. The hearing was destroyed in all cases, the vertigo was relieved in seven but recurred repeatedly in two. The tinnitus improved in five cases and was unchanged in four. Of the two cases with recurrent attacks of vertigo one had a vestibular nerve section done elsewhere six years later because of recurring dizziness usually preceding menstrual periods with reported improvement. The second case with recurrent dizzy spells was reoperated upon and exenteration of the horizontal and superior canal ampullas and contents of the vestibule done.

Ten cases were operated upon (Table 3) by making a small fenestra at the ampullated end of the horizontal canal and ablating the membranous canal with its ampulla (Cawthorne). The ossicles have been left undisturbed in most cases. The hearing was destroyed completely in all cases. The vertigo was relieved in nine and recurred in one case. The tinnitus was relieved in three, improved in three and unchanged in four cases.

The case in which dizzy spells recurred was reoperated upon after three months by exenteration of horizontal and superior canal ampullas and contents of the vestibule.

Four cases were operated upon (Table 4) by making a fenestra into both the horizontal and superior semicircular canal ampullas and enlarging these so as to form a single broad opening leading into the vestibule. The membranous structures in the vestibule and in both semicircular canals were ablated and destroyed as far as could be reached with suitable hooks, forceps and dental excavators. The results have up to the present been uniformly good. Hearing has been totally destroyed and the attacks of vertigo successfully prevented and the tinnitus relieved in all four. The follow-up time has been from six months to two years.

DISCUSSION

Experiments done in this laboratory[17, 18] have demonstrated that in the monkey and cat drainage or destruction of the saccus endolymphaticus along with nearly all of its duct was followed by prompt healing without histological evidence of any disturbance of the inner ear. It seemed therefore obvious that to open the saccus in the human could provide drainage for a very limited time at best and was not a logical procedure for prevention of the dizzy spells in idiopathic labyrinthine hydrops of Ménière's disease.

Simple decompression of the perilymphatic space was done only in one case since there was no good reason to expect this operation to relieve hydrops of the endolymphatic spaces.

The attempt to prevent attacks of dizziness and preserve hearing by destroying the canals at the maximum distance from the vestibule was followed by complete loss of hearing in the one case in which it was tried and was abandoned.

Attempts to destroy or damage the vestibular nerve within the vestibule with the barbed dental broach were followed by complete loss of hearing in all cases but only temporary loss of vestibular function. Despite the return of vestibular function three cases considered themselves relieved of dizziness while the fourth had recurrences.

From this experience it was apparent that to prevent attacks of dizziness consistently a more radical destructive procedure was necessary. It was also obvious that auditory function could not be saved by these methods.

The coagulation technique (Day's operation) failed to preserve hearing in any of the ten cases in which it was tried by us. The dizziness recurred in two. This may have been because of the use of inadequate current in the effort to preserve hearing. It also became apparent that the alleviation of tinnitus was an important objective of any operative procedure and that a more radical destruction of both vestibular and auditory sense organs might be expected to give more consistently good results. Having observed one case operated upon elsewhere with a complete facial paralysis after the use of the coagulating current provided definite evidence that the method was not devoid of risk.

The method of Cawthorne was successful in all but one of our cases in preventing dizzy spells. The effect on tinnitus was however less satisfactory. While there is no doubt that the ablation of one membranous canal with its ampulla prevents further dizzy spells with rare exceptions, the desire to obtain greater relief from tinnitus and at the same time insure complete destruction of the vestibular sense organs prompted the use of a more radical procedure.

The exenteration of the ampullas of both the superior and horizontal canals and the contents of the vestibule is carried out by making a window into both ampullas and enlarging them to create one common opening into the vestibule. The membranous structures are then picked out with hooks and fine forceps. Bone chips are placed in the vestibule to promote filling of the space by osteogenesis.

Removal of the incus facilitates the approach to the vestibule but the malleus and annulus tympanicus have not been removed. The endaural approach has been used in these cases but a postauricular approach would be equally satisfactory. The wound is closed, leaving a small pack in the external meatus for two days to avoid possible narrowing.

A destructive operation on the labyrinth has proven to be a safe procedure but as a therapeutic measure it has limitations. There are certain factors which should be considered in selection of cases. The method is unsuitable for most bilateral cases. The presence of useful unaided hearing in the ear is considered to be a contraindication. The longer the duration of the disease the less likely is it to become bilateral, and the greater the loss of function the less will be the postoperative reaction.

While most individuals develop adequate compensation for the loss of one labyrinth within a few months there are some who continue to have dizziness on quick movements for much longer periods. As a result of these limitations we have found that the incidence in which the operation is clearly indicated is probably well within ten per cent.

SUMMARY

All cases in which labyrinthine surgery has been used in treatment of Ménière's disease or labyrinthine hydrops in the University of Chicago Clinics since 1940 have been reviewed. Surgery has been considered advisable in less than ten per cent.

The various surgical methods designed to prevent attacks of vertigo by destroying vestibular function while preserving the hearing have not proven satisfactory, because of occasional recurrences of vertigo, consistent failure to preserve hearing and frequent persistence of severe tinnitus. As a result of this experience a more radical surgical destruction of the labyrinth has been used in four recent cases with satisfactory results. This consists in exenteration of the ampullary ends of the horizontal and superior vertical canals and through the common opening thus made removal or destruction of the contents of the vestibule.

950 EAST 59TH ST.

REFERENCES

1. Lindsay, J. R.: Labyrinthine Surgery for Ménière's Disease, Tr. Am. Acad. Ophth. 53:527, 1949.

2. Flett, R. L.: Surgical Treatment of the Hydrops of the Labyrinth. The Portmann Operation, Laryngoscope 62:556, 1952.

3. Adams, W. S.: Follow-up of 12 Years Previous Portmann Operation, Rev. Laryng. 71:295, 1950.

4. Barber, H. D. and Ireland, P. E.: Surgical Treatment of Hydrops of the Labyrinth, Laryngoscope 62:566, 1952.

5. Green, R. E. and Douglas, C. C.: Intracranial Division of the VIIIth Nerve for Ménière's Disease, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 60:610, 1951.

6. Lathrop, F. D.: Surgery of Ménière's Disease, Surg. Clin. North Am. 31:929, 1951.

7. Castellano, F.: Ménière's Disease and Its Surgical Treatment, 300 Cases, J. Neurosurg. 8:173, 1950.

8. Day, K. C.: Surgical Treatment of Hydrops of the Labyrinth, Laryngoscope 62:547, 1952.

9. Altman, F.: Surgical Treatment of Ménière's Disease, Laryngoscope 59: 1045, 1949.

10. Passe, E. R. G. and Seymour, J. S.: Ménière's Syndrome, Successful Treatment by Surgery on the Sympathetic, B. M. J. 2:812, 1948.

11. Passe, E. R. G.: Surgical Therapy in Relation to Ménière's Disease, Nerve Deafness and Tinnitus, Acta Oto-laryng. 42:133, 1952.

12. Frenckner, P.: Viewpoints of Operative Treatment in Ménière's Disease, A. M. A. Arch. Otolaryng. 155:420, 1952.

13. Lempert, Julius: Lempert Decompression Operation for Hydrops of the Endolymphatic Labyrinth in Ménière's Disease, Arch. Otolaryng. 47:551, 1948.

14. Rosen, S.: Surgery in Ménière's Disease, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 60:657, 1951.

15. Cawthorne, T. W.: Pathology and Surgical Treatment of Labyrinthine Vertigo of Noninfective Origin. Hunterian Lecture, Ann. Roy. Coll. Surg. England 4:342, 1949.

16. Goodyear, H. M.: Ménière's Disease. Simplified Surgical Management, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 63:261, 1954.

17. Lindsay, J. R.: Effect of Obliteration of the Endolymphatic Sac and Duct in the Monkey, A. M. A. Arch. Otolaryng. 45:1-13 (Jan.) 1947.

18. Lindsay, J. R., Schuknecht, H. F., Neff, W. D. and Kimura, R. S.: Obliteration of the Endolymphatic Sac and the Cochlear Aqueduct, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 61:697-717 (Sept.) 1952.

THE EVOLUTION OF MODERN OFFICE AUDIOMETRY

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In the past ten years, the testing of hearing has advanced so much that it would seem wise to relate it to that which has transpired previously and to that which is likely to follow. It would seem doubly wise because there is confusion in the minds of many about the use of some of the recently developed tests and their value in office practice. This is particularly true of speech audiometry for, in this area, there is reason for this confusion as will be shown later. This discussion will not itemize all the events which have brought us to the present status, nor will it detail the specifics of complicated tests. Rather, it will attempt to orient any who may be puzzled by the present picture.

Undoubtedly the first hearing test ever performed was one which employed the human voice. Whispered and spoken voice tests for years stood at the top of the list of hearing tests. They are still used by some individuals and official agencies even though their fallacies have been pointed out by many, including Glorig.[1] Tuning forks have been employed for many years. They became useful following the tests devised by Weber, Rinne, and Schwabach and are still valuable. Testing hearing by a watch tick was in vogue for years but its general use was discontinued when it was realized it only tested hearing in the range of the frequencies of the particular watch used. The watch test is still used by some examiners for only the day before this paper was begun, the author was called by an insurance official to evaluate the pre-employment hearing test of a claimant as recorded by a watch test expressed in fractions! Of course, it was impossible to place any value on the test. What price progress!

It might interest some of the younger men in the specialty of otolaryngology to know that less than thirty years ago, a hearing test, for an unequal bilateral conductive loss, was recorded at the Massachusetts Eye and Ear Infirmary, as follows:

R		L
5/20	Whispered Voice	10/20
15/30	Spoken Voice	25/30
256	Low tones	128
10,000	High Tones	13,000
10/35	Rinne	20/35
←	Weber	

(No audiometric picture. The institution possessed a 2A Western Electric Audiometer but it was not used for routine testing.)

In the voice tests above the denominator represented the limits (in feet) of normal hearing and the numerator recorded the distance (in feet) at which the patient responded. The "low tones" were the lowest pitched tuning fork heard and the "high tones" were the responses to a Galton whistle which was calibrated to read up to 22,000 double vibrations per second! The Rinne test, performed with the 512 dv fork, was timed in seconds and only this and the Weber test are familiar to the younger otolaryngologist. It should be noted that the Weber test followed the Rinne whereas the reverse order should have been followed. Undoubtedly, under this order, many tests were incorrectly interpreted. This is true because masking was not employed. To state that masking was not used is not exactly correct as it was a routine procedure for the patient to place one of his fifth fingers in the ear opposite to the one being tested.

It must not be assumed from the foregoing that this represented poor otologic practice. Far from it. There were some really great otologists on the staff of the Massachusetts Eye and Ear Infirmary then. It clearly shows, however, that it was impossible to know as much about the hearing of a patient then as we are able to discover now.

It was not until the nineteen-thirties that the use of the pure tone audiometer became general. This came about in the latter half of that decade, so it is less than twenty years since pure tone audiometry became popular. It was not a burning desire on the part of many otolaryngologists to change to newer and better testing of hearing which brought about this increased ownership of audiometers. Rather, it was the fact that several manufacturers of audiometers had constructed models which were available at a reasonable cost, and many doctors bought them because of sales pressure. ("Dr. Jones

and Dr. Smith have purchased one. You can't afford to be without an audiometer." This was a frequently used sales talk.)

The change was a sweeping one but it was not complete nor thorough. The emphasis was placed on tests by air conduction and only a few began at once to do bone conduction tests. Little or nothing was mentioned in those days of the need for masking, particularly in bone conduction audiometry. Another rather serious error crept in. This new means furnished precision testing contrasted to the older methods and the audiogram was looked on as an accurate appraisal of hearing without too much thought being given to the fact that the twister of the dials might not know too much about his machine and that the testee might give erroneous responses. It is now known that it is wise to employ confirmatory tests (especially in patients with mixed hearing losses) to appraise the hearing function of an ear but it was not generally realized then.

It was not until the early nineteen-forties that the general use of bone conduction audiometry came to pass. Along with this, masking began to be used. There was little recognition then of the fact that the type of masking noise available in commercial audiometers (saw-tooth noise) is not too satisfactory. In most instances, too, there were no means by which the tester could know the intensity of the masking noise employed. It should be noted that even today many teachers do not require students to use masking when they are doing bone conduction tests with tuning forks, while these same teachers make masking during audiometric bone conduction studies obligatory.

World War II brought about many changes. Not the least of these was a marked advance in the field of electronics. This advance made possible the discovery and employment of many additional methods for testing hearing. Some of these advances include two channel audiometers, speech audiometry, tests for recruitment, testing by psychogalvanic skin resistance, testing by electroencephalography, the Békésy audiometer, and such special tests as delayed speech feed-back, difference limen, the Doerfler-Stewart test, etc., etc. What do these advances mean? Do they mean that the average otolaryngologist should employ all of these tests on every patient whose hearing is impaired? That is not necessary, of course. A reasonable view is this, that these tests are available and the more complicated ones should be used by those interested in problem cases. Otolaryngologists should be informed about them and be able to determine when they should be instituted and when they are not necessary. Several of the less complicated tests should be part of the diagnostic armamentarium of every otolaryngologist. This is particularly true of speech audiometry. Why?

Earlier in this paper mention was made of the fact that today it is recognized that in testing hearing one should employ more than one method as a check to eliminate possible errors. Testing hearing by speech, though it yields information about the ability to hear words and sentences, is also valuable as a check on the results of pure tone audiometry both by air and bone. This is the opinion of many who use speech audiometry. Its importance was emphasized in October 1952, by a statement of the "Committee of Eight." This Committee has equal representation from the American Medical Association and the American Academy of Ophthalmology and Otolaryngology. It is charged with the responsibility of a realistic revision of the present methods of reporting the percentage of hearing loss in compensation cases. After due deliberation, it announced to the American Academy, then in session, that in their opinion speech audiometry would be a basic part of any new method devised by them. This was the pronouncement of the committee despite the recognition of difficulties which would be encountered while testing the hearing of patients with language deficiencies.

What is the present status of speech audiometry in this country? One has to sketch briefly its history to evaluate its status. Whispered and spoken voice tests have been mentioned. Decreasing intensity numbers tests were in use in school audiometers for some years before World War II. Prior to the early nineteen forties, several individuals arranged and used their own speech testing material. All of this made some contribution to the development of speech audiometry as we know it today. The work done at the Psychoacoustic Laboratory at Harvard during World War II, however, gave it a sounder scientific basis. Its first enthusiastic clinical application was by Walsh and Silverman[2] who used it for further determination of the suitability of candidates for the fenestration operation. While speech audiometry is not yet in general use in this country, it is probably safe to say that most fenestrators today use it as a preoperative test for suitability. This is because it is apparently a reliable check on pure tone bone conduction readings and permits a reasonably accurate appraisal of the amount of cochlear reserve present, something which every fenestrator wishes to know. It is information which a surgeon needs when talking to a patient about the possible outcome of his fenestration operation.

There are at least two important reasons why speech audiometry is not commonly used despite its demonstrated value. These are, 1) the lack of standards and 2) the time required for such testing. The first of these, the lack of standards, needs further explanation

for it is the prime reason for the confusion in the minds of many relative to the use of speech audiometry in office practice.

The first recorded speech tests in this country which had more than local use were on records processed by reliable persons. This was fortunate but their use by several hearing centers and some individuals brought to light the fact that even with adequate amplification they lacked one important feature, an entirely satisfactory discrimination test. Patients with normal hearing, tested by these records, often failed to obtain a 100 per cent discrimination score. Obviously normal hearing patients should have such a score. Inasmuch as the discrimination test is one of the essential values of speech audiometry this was an important lack.

After more than a year this fault was recognized and later new records which were supposed to correct this fault were sold by the same individuals. The new records over-corrected the fault. The recorded material was easier to understand and too many patients were able to obtain a 100 per cent score. In other words, some patients with a moderate degree of perceptive loss obtained a perfect score. Further, patients with marked perceptive losses received relatively high percentage scores. The spread between these scores and a 100 per cent score was not sufficient to adequately separate different degrees of perceptive loss. With this new scale of values a change of only a few percentage points represented too widespread a difference in the amount of perceptive loss present. The records failed to establish satisfactorily what they were supposed to do; furnish the examiner with a reasonable appraisal of the degree of perceptive loss in the patient tested.

To date no satisfactory recorded material which has the sanction of reliable parties has been made available to replace the records mentioned above. This is unsatisfactory and is a significant reason why there is confusion in the minds of many about the present status of speech testing. This confusion prompts a question. "How can one institute speech audiometric testing in the office when there are no sanctioned recorded materials?" The answer to this question is that there are those who have instituted it and who would not be without it. "With what means?" is the next question. Some are using the records mentioned above, some are using other commercially available records, others have recorded their own material either on records or on tape and still others are using live voice either in a one-room or a two-room set-up.[3] Each of these has established his own standards! While this is satisfactory from the standpoint of the individual, wrong impressions may arise when the results of one are compared to those of another. However, systems which will perform

within certain limits can be practical and the results obtained from
their use can be compared with the results obtained from others.
One such limit should be that when testing patients with conductive
hearing losses, one can obtain a speech reception threshold (a 50 per
cent correct interpretation of the test material) within 5 decibels of
the average of the pure tone losses at the three speech frequencies, 500,
1,000, and 2,000 dv.   (Since there are several types of material in
use for obtaining speech reception thresholds and some of these are
more difficult to understand than others, the zero reference level of
individual systems must vary, according to the speech tests employed,
if the above limit or requirement is to be met.)   Another requirement
should be that, at an intensity of 25 decibels above speech reception
threshold (SRT), individuals with a pure conductive loss should reg-
ister a discrimination score of 90 per cent or better.   Theoretically,
if sufficient intensity is employed, every such patient eventually
should have a 100 per cent discrimination score.

The limits for patients with perceptive losses cannot be as defi-
nite as those for conductive losses, simply because the ability to un-
derstand speech is not always of the same order as the ability to hear
pure tones.   With many perceptive losses the SRT will approximate
the average of the pure tone losses at the three speech frequencies.
But there are other perceptive losses with a marked discrepancy be-
tween the pure tone average and an obtainable SRT.   This discrep-
ancy may be considerable if the loss is marked in the lower frequencies
("helicotrema hearing loss").   Occasionally such patients with pure
tone audiograms showing a flat or even a rising curve, having losses in
the order of only 50-60 db, may be unable to respond correctly, no
matter what amplification is employed, to any of the speech reception
threshold material used (Fig. 1).

Thus discrimination scores from patients with perceptive losses
do not conform to set limitations, i.e., so much pure tone loss, so much
percentage loss.   It is now generally agreed that the greater the dam-
age in the organ of Corti, the lower the discrimination score will be.
So it is practical to say that in a mixed loss (perceptive plus con-
ductive where the perceptive element does not override the conduc-
tive element), at 25 db above SRT, the probable discrimination score
will be in the higher percentages (above 70 per cent but below 90
per cent. Discrimination scores obtained from patients with perceptive
loss only, or predominately perceptive, will vary from these higher
percentages (70 per cent to 90 per cent) to zero.   The percentage
obtained will depend upon the amount of cochlear damage present
(especially toward the region of the helicotrema) and not upon the
pure tone response.   Testing at several intensities greater than 25 db
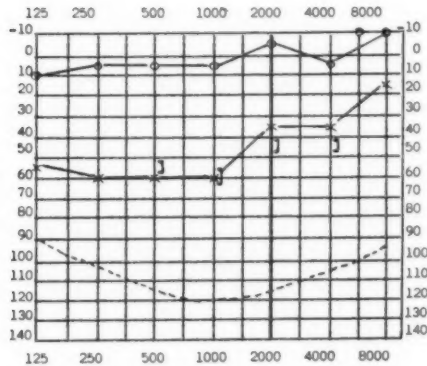
Fig. 1.—Mr. L. J., sudden loss of hearing A. S.   No S.R.T. obtainable
in left ear.   Discrimination score 0 per cent.

above SRT may be employed.   In certain instances, notably in true
Ménière's disease, the discrimination score will decrease as the intensity
increases.   A speech audiometric system which will yield such results
is practical and will furnish the examiner with invaluable informa-
tion.   Such a system is possible for any practitioner, provided good
materials are used (approved tests and equipment) under proper
testing conditions.

The author employs live voice in a one-room set-up and is able
to obtain consistent results along the lines noted above.   Live voice
in a one-room set-up does not permit the use of speech audiometry
for slight hearing losses, for such patients, even with bilateral receivers
on their ears, can hear the live voice outside the system to a degree that
precludes conclusive results.   In the author's testing room an expensive
speech audiometer with turn-table, built by an extremely competent
electronics engineer, stands idle awaiting the day when satisfactory
standardized material becomes available.

What of the future?   When will standardized recorded ma-
terial become available?   The Committee of Eight, mentioned above,
knew, when it recommended speech audiometry as a basic factor in
reporting percentage loss of hearing, that there were no standard
recorded materials available.   It realized if a new and approved method
of reporting percentage hearing loss was to be recommended that a
requirement would be the availability of approved testing material.
Steps have been taken to make this possible.   Through a grant from
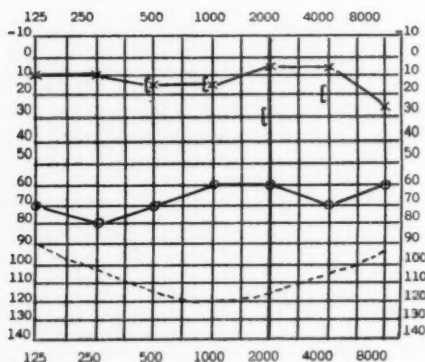
Fig. 2.—Mrs. J. R., bleeding from A. D. following head injury. S.R.T.
A.D. 60 per cent. Discrimination score, 25 db above S.R.T. 96 per cent.

the National Institute of Health, studies have been under way for
over a year and will continue for some time. The attempt is being
made to create tests which can be validated by other hearing exam-
inations and known hearing experiences (difficulty in group con-
versation, etc.). It is estimated that it will be two years at least before
such material can be properly assembled, recorded, and made avail-
able commercially. This means that an otolaryngologist who has not
yet inaugurated speech testing in his office must either establish a
temporary (but practical) system now, or wait at least two years.

One does not sense the value of speech audiometry until one
practices it for a period. After some experience with it, one won-
ders how he got along without it. A colleague will ask an opinion
about a pure tone audiogram and an immediate reaction will be an
inquiry as to the results of speech tests. This opening of a new
horizon is like the change from whispered and spoken voice and tun-
ing fork tests, to pure tone testing. No one would gladly submit
to a return of those days. An actual illustration of the confirming
value of speech audiometry was demonstrated recently when a cer-
tain patient was tested. Eighteen months previous to her examina-
tion, she had a skull fracture and a bleeding ear which resulted in a
one-sided hearing loss. Pure tone audiometry (bone and air) showed
a hearing loss on that side with a marked conductive element in it.
It seemed something must be wrong with the testing. But speech
audiometry also showed this conductive element. The discrimination
score at 25 db above SRT was 96 per cent (Fig. 2). Investigation
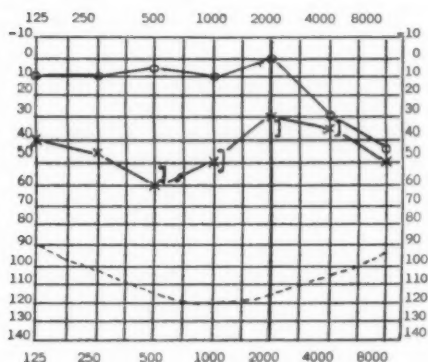
Fig. 3.—Mrs. G. P., severe attacks of dizziness for seven years. Complaining bitterly about the effect of loud sounds and distortion in the left ear. S.R.T. A.S. 55 db. Discrimination score 20 per cent. (No useful

revealed there had been a dislocation of the ossicular chain. This was not a simple instance of concussion or fracture.

Undoubtedly one of the biggest objections to be raised against the establishment of speech audiometry as an office procedure will be the time involved in such testing. Inasmuch as pure tone testing will probably never be replaced by speech audiometry it is obvious that anyone who adds the latter to his routine will have to allow more time for his total testing procedure. If speech testing then is to become universal it must not be too time consuming. When Walsh and Silverman first proposed complete prefenestration hearing surveys, they estimated their tests required from three to four hours to perform. Practical speech testing, however, can be done in a very few minutes except in problem cases. The actual number of minutes needed will vary according to the different ways speech samples may be presented. Future studies should be directed toward evolving testing procedures which, without wasting time, will yield maximum information. Recorded material can be administered by an assistant, thus saving the otolaryngologist's time. If live voice is used the examiner himself had best administer the tests unless the one who is assigned the task has had experience and has proven worth and integrity.

There are practical aspects to all of this. Prefenestration evaluation of hearing has been mentioned. Speech audiometry is equally valuable for testing patients who may be candidates for the Day

operation (surgical obliteration of the labyrinth). Such a patient may have a pure tone audiogram which is reasonable, when in reality he has little or no useful hearing. Speech tests bring this out as no other test does (Fig. 3). In the light of such information, one can proceed with an easy conscience to destroy an ear so involved.

Another practical application is the studied advice one can pass on to patients who wish to know whether or not they can successfully wear a hearing aid. A patient who has a moderately poor discrimmination score may be able to wear an aid successfully, but he should be counselled that one should not be purchased till either a hearing aid evaluation has been made at a hearing center, or a trial period has established its wearability. A patient with a discrimination score below 50 per cent, according to the practical requirements above, cannot wear an aid unless he is one of those unusual individuals who obtains considerable information through the tactile sense (these are almost always young persons). Still further, a patient who has a low level of uncomfortable loudness for speech, one who has uncomfortable loudness at only 15-20 db above his SRT, will experience great difficulty in wearing an aid for there is no machine made which can buffer out sounds, so the remainder are confined to this narrow band. Incidentally, uncomfortable loudness studies are informative when testing routinely (when hearing aid selection is not an immediate consideration). It is another test which should be added to discrimination scores and recruitment studies. These three are interrelated and when all are available to the examiner, he can better appreciate the difficulties a patient may have in listening to and understanding conversation. The author routinely tests for uncomfortable loudness in all patients who have a perceptive element in their hearing loss. It takes less than a minute to perform.

Still another practical application and a very important one is the insistence which, in the near future, industrial organizations and insurance companies will place on complete hearing tests. The peak load of testing for hearing loss caused by industrial noise has not arrived, but it is probably just around the corner. The otolaryngologists who are well equipped and experienced in testing will probably get the bulk of this work. If the new method for reporting percentage loss includes the results obtained from speech audiometry, as present deliberations of the Committee of Eight indicate, then only those who have had some experience with it will be asked to do such testing.

Guild[4] at the end of the nineteen forties sounded a conservative note about the relative value of the hearing tests which had been developed in the previous decade. Among the statements made at

that time he said "The conclusion is inescapable that the knowledge needed to interpret hearing tests intelligently has not increased as rapidly as have testing techniques." He also stated that many of the newer tests do not aid in making a diagnosis and that the cause of hearing loss can often be determined by relatively simple tests interpreted in the light of the history and the physical examination. He added that many of the newer tests should be reserved for those engaged in research, and were not suitable for office practice. All of this is true. It is the reason why this evaluation of present day audiometry does not detail the mechanics or value of the PGSR test, the Békèsy audiometer, testing by electroencephalography, etc. Speech audiometry does not fall into this group. It cannot, in the majority of instances, help establish a diagnosis as well as pure tone audiometry and tuning forks. But as a result of experience with speech testing over the past ten years, it is now realized that pure tone audiometry does not measure the capacity to hear and understand speech to the degree that is needed to properly advise patients who have an irreversible hearing loss. The otologist should have a reasonable idea of a patient's ability to receive oral communication before attempting to outline treatment either medical, surgical or rehabilitative.

SUMMARY

An attempt has been made to evaluate speech audiometry in the light of the past and the present status of hearing tests. Much of what has been outlined has been described in detail by Walsh,[5] Cawthorne[6] and many others.

It would be foolish to predict what tests may be evolved in the future. We have come a long way from the hearing tests of a quarter of a century ago. There are so many tests and techniques available today, it will take some years before we will know the relative value of each. From the testimony of many, speech audiometry is one of the significant methods of testing hearing and indications are that it may soon become an integral part of all hearing evaluations.

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REFERENCES

1. Glorig, A.: Hearing Evaluation by Low Conversational Voice Tests, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 58:394-402 (June) 1949.

2. Silverman, S. R., Thurlow, W. R., Davis, H. and Walsh, T. E.: Improvements in the Social Adequacy of Hearing Following the Fenestration Operation, Laryngoscope 58:607, 1948.

3. Work, W. P.: Two Room Audiometry Set-up for the Otologist's Office, Laryngoscope 59:454-468 (May) 1949.

4. Guild, S.: Hearing Tests and Testing, Laryngoscope 59:674-679 (June) 1949.

5. Walsh, T. E.: Speech Audiometry, J. Laryng. and Oto. 67:119-127 (Mar.) 1953.

6. Cawthorne, T. E. and Harvey, R. M.: A Comparison Between Hearing for Pure Tone and for Speech, J. Laryn. and Oto. 67:233-247 (May) 1953.

In the laboratory, 1939.   PHOTO WERNER MUELLER

XII

SURGICAL APPROACHES TO DEEP NECK INFECTION

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In 1931, at a meeting of the American Academy of Ophthalmology and Otolaryngology, I mentioned to Dr. Mosher that I had treated a number of cases of deep neck infection over a period of a few years. He was immediately interested, and asked me to describe my experiences in a paper on the subject. That paper was written, and read at the meeting of the Academy the next year, 1932.[1] He had for some time sensed the importance of the subject to the otolaryngologist and to the profession as a whole. To him is due the major credit of interesting the otolaryngologist in the surgery of the neck. As a result there followed a long series of papers by a number of writers.

In 1920 he[2] first called attention to the relationship between neck abscess and thrombophlebitis of the internal jugular vein. His paper in 1929[3] was one of the most important publications of his entire career. I have in the past characterized it as being the most important surgical procedure in the neck since tracheotomy. His drawing illustrating the T-shaped incision was made intentionally grotesque. I am sorry that I neglected to ask him his reason, but I suspect that it was the result of his keen teaching psychology, coupled with a streak of humor. His surgical approach was accurately based on sound anatomic fundamentals. Whatever success I have had in the surgery of deep neck infection is due entirely to this anatomical groundwork. It was my privilege to follow up with some of the clinical applications. Others have made important clinical contributions.[7-31] Previous to that time practically all papers on neck infections were written by general surgeons. In 1908 T. Turner Thomas,[4] a general surgeon, published several articles on Ludwig's angina, which may be considered classics. In 1946 Dr. Mosher quoted from Cheever's Surgery,[5] on the subject of deep cervical abscess; I believe that I am correct in stating that Dr. Mosher was one of the first otolaryngologists, if not the first, to publish a paper on the subject of deep neck infection.

In 1929, when the sulfonamide drugs became available, the beginning of a new era of therapy for infections was born. A few

years later, between 1941 and 1945 while World War II was in prog-
ress, penicillin came into use, soon followed by other antibiotics.[6]
These chemotherapeutic and antibiotic drugs have been remarkably
instrumental in bringing about control of nearly all bacterial infec-
tions, especially the formidable pyogenic group. Their efficacy in the
treatment of syphilis, gonorrhea and tuberculosis is evidence of their
value in the control of infections not caused by bacteria of the pyo-
genic group. These improvements in the control of infectious diseases
have made it necessary to revise and correlate our medical and surgical
treatment. Despite these improvements in the control of infectious
diseases it will probably always be necessary to know something about
surgical drainage of the neck, and it behooves us not to throw away
our scalpels, nor to lose the knowledge of how to use them.

It soon became apparent that these new drugs were not always
harmless, and that sometimes they cause severe toxic and allergic
reactions. These side effects sometimes make it necessary to discon-
tinue administration of the drug in an occasional case. Constant
watchfulness is necessary. We all know of the toxic effects of the
sulfonamides. When penicillin was a new therapeutic agent it was
regarded as having no toxic characteristics. Accordingly the amount
of the dosage varied within wide limits. Later, the repository prepara-
tions received wide popularity because it was possible then to lessen
the frequency of the injections, and avoid the discomfort and loss
of sleep incident to them. Gradually reports of untoward allergic
reactions appeared. Briefly, those that give us most concern are of
two varieties: first, those in which, about the ninth or tenth day,
there appear urticaria and other skin involvements, arthritic edema
and pain, localized superficial edema of skin and mucous membranes,
etc.; and second, those which in a few seconds or minutes cause pa-
tients to lapse suddenly into anaphylactic shock from which some
do not recover. These have been attributed to sensitization by pre-
vious administration of penicillin. There have been a few reports of
fatal anaphylactic shock in patients who have had no history of pre-
vious administration. When a history of recovery from anaphylactic
shock is elicited a skin test for allergy, with even the minutest quantity
of penicillin, should never be done. I know of one case in which this
was done, and the patient went into a more severe state of shock
than the first time, and was saved only by the most heroic measures.
It is impossible to overestimate the danger of such reactions, and
when such reports come from medical examiners who do the autopsies
they must be taken seriously.[32] It has occurred to me that perhaps
it may be possible for an individual to become sensitized by the drink-
ing of milk from cows which had been treated with penicillin. So far
as I know no investigation along these lines has ever been done, but

if sensitization becomes frequent or prevalent it is my humble opinion that such a sequence of events should be thoroughly investigated. The addition of an antihistaminic drug to each injection and in the same syringe can be expected to prevent or minimize some of these reactions. The antihistaminic may be given intravenously. In these severe infections parental medication is still to be considered our main reliance.

As a result of the frequent administration of penicillin for many kinds of bacterial infections, organisms have shown a strong tendency to become resistant to the drug. This applies particularly to the staphylococcus group. It is now common practice to test the resistance of organisms growing on culture media, to the various kinds of antibiotics available for treatment. Those antibiotics showing the greatest bacteriocidal effects are chosen for treatment.

Following the use of antibiotics there has been a remarkable change in the character of the work that the otolaryngologist now performs. Formerly it was chiefly surgical in that in the majority of cases some kind of surgical drainage was necessary to bring about termination of the infection. Surgical drainage is not now so often required, and the general physician and the pediatrist treat many of these cases in an early stage, often bringing about a cure before any complications can get started. Severe suppurative sinusitis and acute suppurative otitis media and mastoiditis are now much more rarely seen and treated by the otolaryngologist, particularly in an early stage. Otitic meningitis and lateral sinus thrombosis are not the common occurrences they once were. Young physicians who are about to choose their future fields of practice from among the many specialties available are not now so frequently seeking residencies in otolaryngology, and hospitals are finding it difficult to obtain young men of ability to serve in the department of otolaryngology. It is indeed a changed specialty. However, in the expressed opinions of many of the leaders of otolaryngology, opportunities for the furtherance of important contributions in the specialty are, and will continue to be, just as numerous and attractive as they ever were, but of a changing nature. New avenues of investigation and accomplishment will be opened up, which we cannot now entirely foresee. Some of these will be medical, as, for instance, biochemical; and some will be surgical, such as tumors, congenital lesions, and even infections.[33] Optimism is entirely justifiable. In fact the future of the subject of virology alone is so vast and undeveloped that future discoveries will influence the science of medicine in its entirety. One of the greatest of these unsolved problems, the common head cold, lies within the field of otolaryngology, and is often referred to as the most com-

mon of all unsolved diseases. The basic physiology of allergy is still
largely conjectural. The numerous causes of the common symptom
called tinnitus will not soon be completely tabulated. Immunology
is still in its infancy. Who can say how much and in what way the
antibiotics will cause alterations in the natural protective mechanisms
of the body? What effect will the elimination of one type of infection
have on the resistance to other types? Will the solution of one prob-
lem beget or reveal another? Knowledge of the endocrine system is
still far from complete. One could cogitate along these lines indefi-
nitely. It will be necessary for the otolaryngologist to keep abreast
of research in general medicine in order not to miss discoveries which
have important bearings in the field of otolaryngology.

Preventive medicine aims to eliminate or reduce the frequency
of infectious diseases, and may be compared to the safety measures
which have been devised to reduce accidents on streets and highways.
If carefully observed they do reduce accidents, but accidents do occur
when these safety measures are ignored; and governmental enforce-
ment agencies must still be maintained. Likewise, surgical agencies
will still be necessary in the field of medicine, and neck infections that
require drainage will still occur.

With this viewpoint in mind it is now my purpose to describe
the surgical approaches to drainage of neck infections.

SURGICAL APPROACHES

Superficial Infections. The surgery applied to drainage of fu-
runcles, carbuncles, and superficial infections resulting from trauma
is not here under consideration.

Infections of the neck may be designated as superficial and deep.
Superficial infections are subcutaneous and are usually related to in-
fection of the superficial groups of lymph nodes in relation with the
superficial cervical fascia. Specifically these nodes are situated chiefly
in the preauricular region, in the upper angle of the occipital triangle
adjacent to and behind the attachment of the sternomastoid muscle,
in the submental triangle, and along the external jugular vein. Ab-
scesses in these nodes are subcutaneous and manifest themselves im-
mediately. Fluctuation is readily elicited, and indications for drain-
age are soon present. This is the only type of neck infection in which
it is justifiable to wait for fluctuation, provided that the sepsis is mild.
There is no justification for the procedure of aspiration by needle
puncture in any type of superficial or deep neck infection. Super-
ficial abscesses present no problem of approach and may be incised
at the point of fluctuation. Deep extensions must not be overlooked.

If lobulated, all soft tissue septa or partitions should be gently broken down with the finger, creating one cavity which may then be lightly packed with some form of gauze, my preference being iodoform gauze. Slow bleeding from granulation tissue and minute veins is thus controlled. Bleeding from larger veins and any arteries should be controlled with ligatures. The incision should be slightly longer, rather than slightly shorter, than necessary for good exposure. When dealing with these superficial abscesses, incisions in a more or less horizontal, or encircling, direction heal with less subsequent distortion than do vertical incisions because they are more nearly parallel with the elastic fibers of the skin. If for any reason it should be advisable to expose the carotid sheath or its contiguous lymph nodes there should be no hesitance in making the incision along the anterior border of the sternomastoid muscle to obtain better exposure. In such circumstances the cosmetic aspects are not paramount. If the abscess has been completely emptied and there is minimal sepsis, manipulations other than for the insertion of drainage material are unwise, and serve only to traumatize unnecessarily. As Mosher has stated, all layers of the cervical fascia stem are from or are connected with the carotid sheath, and therefore localized infections may be searched for through this exposure. A short incision results in a small, deep, funnel-shaped hole in which, because of poor exposure, it is difficult to obtain good visibility and working room. Control of bleeding is also more difficult. A large incision with well loosened and well retracted flaps gives a flat and shallow field, minimizes the difficulties, affords better visibility of bleeding points, and makes for greater safety. Veins are seen before they are accidently injured, and may be leisurely divided between two ligatures. In the neck bleeding must be securely controlled with ligatures. Pressure may not be applied.

PERORAL DRAINAGE

There are several kinds of infection which may be drained by internal, or peroral, incisions.

Retropharyngeal Abscess. Nearly all cases of retropharyngeal abscess may be drained by peroral incision of the posterior pharyngeal wall. With suction at hand the head should be placed in a position lower than the chest, so that pus is prevented from entering the trachea and bronchi. A mouth gag should be inserted and securely held, and a blunt instrument such as the closed blades of suitable size scissors or clamp quickly thrust through the abscess wall and opened just before removal. The mouth gag minimizes swallowing. The end of a mouth suction tube should be in position in the pharynx

during the time this maneuver is being carried out. If there is any difficulty in breathing tracheotomy should be done first. The necessity for a hasty incision is eliminated when the airway is free. When a retropharyngeal abscess has gravitated to the level of the larynx there is always more or less impairment of breathing. If an exact diagnosis of the location of the abscess cannot be made by inspection, and sometimes it cannot, a lateral neck radiograph with soft parts exposure time is immediately indicated for diagnosis. If breathing is severely impaired the tracheotomy should be done first, after which the lateral neck radiograph may be leisurely obtained. These cases are most commonly found in infants and young children and, in the severe cases, life sometimes hangs by a slender thread. If sepsis persists after peroral incision, whether or not pus was evacuated, external drainage must be considered. Unwise attempts to visualize or palpate the pharyngeal cavity in a patient with marked impairment of the airway may result in asphyxia. After tracheotomy both patient and doctor may breath freely.

Sublingual Incisions. Infections sometimes localize in the floor of the mouth.[14] These usually follow dental pathology, dental surgery, and infections of the salivary glands and their ducts, with or without calculi. It is sometimes possible to drain them by intraoral incisions. When working in the floor of the mouth, incisions should avoid the lingual vessels and the lingual nerve in the posterolateral region on each side. In the anterior part immediately behind the incisor teeth there are no important vessels or nerves. Posteriorly these vessels and nerves lie on the tongue side of the submaxillary duct. The lingual nerve lies to the lateral side of the duct, and courses under the posterior end of the duct near where it joins the gland, and from there goes medially into the tongue. Avoid working beneath the posterior third of the salivary duct. A thrust with a blunt artery clamp is safer than deep incisions with the scalpel. If superficial incisions are inadequate, external drainage in the suprahyoid region should be the approach. For severe infections better drainage is obtained through the external approach. When there is marked trismus the external approach is a necessity.

Dentoalveolar. Sometimes infections from the molar and premolar regions of the mandible rupture spontaneously through the inner plate of the mandible, and pus appears in the mouth through perforations in the soft parts, or gums. These are most frequently seen and treated by the dental or oral surgeon. Drainage may be quite successfully obtained through a fistula, and the underlying dental or alveolar infection may be dealt with at the same time. When located in the region of the third molar tooth the homolateral sides

of the soft palate and tongue base are involved. A prominent diagnostic sign in such cases is extreme pain and discomfort when a tongue depressor is used.[24] If the third molar tooth is still present it is often almost completely hidden by the edematous and swollen contiguous soft parts. When symptoms of pain and discomfort suddenly and spontaneously subside one should look for a fistula and beads of exuding pus in the gum tissues which are in relationship to the tooth. It should be remembered that in these sublingual and dentoalveolar infections, there is sometimes a pharyngomaxillary infection which was introduced by the needle when blocking the mandibular nerve. Through the external submaxillary fossa approach all of these infected areas may be simultaneously drained. If the sublingual involvement is predominantly in the midline, and there is marked trismus, it should be reached by the external suprahyoid midline approach which will be described later.

Peritonsillar Abscess. A large proportion of peritonsillar abscesses, perhaps a majority, may be drained by peroral incision through the upper third or half of the anterior pillar. Such cases are often treated and drained by the general physician, and usually conclusively. Many rupture spontaneously. Usually no anesthesia, either local or general, is applied. Local injection into such inflamed tissues is contraindicated because it would increase the inflammation greatly, and because it would be as painful as an incision. In cases in which the abscess localizes behind or at the lower pole of the tonsil, general anesthesia is justifiable in selected cases in order to remove the tonsil from its bed in search for the abscess. The possibility of the abscess having extended into the pharyngomaxillary space should be considered, in which event the external approach should be used. A small abscess below and behind the tonsil cannot be reached through the usual incision in the upper part of the anterior pillar. Occasionally I have drained such an abscess by removing the tonsil. The pillars are liberated by incision at the margins, and the tonsil is liberated by blunt dissection with a small piece of rolled gauze in the grasp of a clamp. I have encountered no dangerous bleeding in such operations, and healing is as uneventful as after ordinary tonsillectomy. Infection from such a source may invade the pharyngomaxillary space, or may gravitate downward along the visceral fascia.[17] Deep neck infection, therefore, may sometimes be prevented by timely tonsillectomy. In such cases it has been my custom to remove the other tonsil at a later time. When dyspnea is already present restoration of the airway by tracheotomy is the primary consideration, after which the external approach should be used, one drain in the pharyngomaxillary space, and one along the visceral fascia adjacent to the inferior constrictor muscle.

A type of drainage material which I have found very satisfactory
is that shown in the accompanying drawing.

In the neck, where complete immobilization is not attainable
drainage tubes are apt to become displaced and sometimes extruded
even when secured to the skin margins by suture. A drain such as
is here shown and described is not easily displaced nor extruded be-
cause the soft tissues protrude into the loop at the end, and tend to
hold the drain in position. The drain may be sutured to the skin,
but I have not found this to be necessary. Ordinary Penrose tubing
is satisfactory for the carotid sheath region.

INFECTION OF THE CERVICAL LYMPHATICS

The superficial groups of cervical nodes lie between the super-
ficial cervical fascia and the superficial layer of the deep fascia, and
are therefore in close proximity to the overlying skin. Drainage of
abscesses involving these nodes has already been described. The deep
chains are arranged in certain more or less definite group chains; some
chains horizontal, others vertical or oblique, and others irregular.
They are situated at all fascial depths, and all layers of the deep fascia
are plentifully studded with lymphatics. Clinically the deep chains
of nodes of both the head and the neck should receive combined con-
sideration. The same group may be located in the head and in the
neck, as, for example, the parotid and the preauricular lymphatics.
High up in the retropharyngeal space there are several lymph nodes
which take drainage from the nose and nasopharynx. When these
suppurate a retropharyngeal abscess is the result. In a more or less
horizontal line from the occiput to the chin, there are several groups
of nodes, namely, the suboccipital, the posterior auricular or mastoid,
the preauricular and parotid, the submaxillary and the submental.
In the posterior cervical triangle there are nodes in close series with
the sheath nodes. They extend from below the occipital and mastoid
bones to the clavicle and the scapula. Those in the parotid group are
found on the fascia of the gland, and some are enmeshed in the parotid
gland structure. Swelling of the parotid gland region closely resem-
bling mumps, may be caused by inflammatory swelling of these lymph
nodes alone. Receiving the drainage from all lymphatics of the head
and neck is the large deep chain situated along the carotid sheath be-
neath the sternomastoid muscle, and arranged in a more or less vertical
direction. This is the most important lymphatic group in the neck.
Those nodes along the carotid sheath which lie above the inferior belly
of the omohyoid muscle are termed the upper or superior deep cer-
vical nodes. Those lying below this muscle belly are termed the lower
or inferior deep cervical nodes. They are best exposed for drainage

or excision through an incision along the anterior border of the sterno-
mastoid muscle. If a large necrotic cavity is exposed it usually suffices
to pack the cavity lightly with gauze. If the nodes are intensely
inflamed, swollen, and adherent to the carotid sheath it is advisable
to excise them in order to obtain good visualization of the sheath. If
symptoms and signs of severe sepsis and blood stream infection are
present it is advisable to ligate or resect the internal jugular vein, and
to place a soft rubber Penrose tube drain on the sheath. If the lower
deep nodes are also involved it is sometimes advantageous to divide
the inferior belly of the omohyoid muscle to get better exposure.
Severe infection of these nodes may be the cause of carotid sheath
infection with or without blood stream infection.

REGIONAL SURGICAL APPROACHES

Prevertebral Fascia Infection. a) Peroral incision of posterior
pharyngeal wall (described previously); b) incision along anterior
border of sternomastoid muscle, as described by Dean.

The approach for external drainage of retropharyngeal abscess
has been described by Dean. The incision is made along the anterior
border of the sternomastoid muscle, exposing the carotid sheath.
The muscle and sheath are retracted laterally. Usually the attack is
made above or at the level of the larynx lateral to the inferior con-
strictor muscle. The superior thyroid vessels and superior laryngeal
nerve should be identified and retracted medially. The hypoglossal
nerve and the lingual and facial vessels should also be identified and
avoided. The abscess will most often be found at the level of the
hypopharynx. After its prominence is located a blunt instrument
such as a closed clamp is thrust into it, and the opening enlarged by
separating the jaws of the clamp. The opening is made between the
sheath which lies laterally and the inferior constrictor which lies
medially. If the abscess has descended toward the chest the approach
must be extended downward along the viscera as described for cer-
vical mediastinotomy. If dyspnea is not severe enough to prevent
inspection of the throat, and there is no danger of asphyxia, this ex-
ternal approach might make it possible to avoid tracheotomy. Gen-
eral anesthesia of any kind is contraindicated just as in any other
condition which is associated with dyspnea. In cases in which severe
dyspnea is present tracheotomy should be done first to establish a
good airway. After tracheotomy, general anesthesia may be admin-
istered.

Pharyngomaxillary Space Infection. a) Submaxillary fossa ap-
proach as recommended by Mosher. b) Approach beneath the angle
of the jaw directly. c) Approach through the posterior triangle.

Fig. 1.—Mosher's original drawing showing the T-shaped incision for
the submaxillary fossa approach to the pharyngomaxillary fossa.

As previously stated, the submaxillary fossa approach as described
by Mosher in 1929 is, I believe, the most important procedure per-
taining to emergency head and neck surgery since the advent of
tracheotomy.

The detailed description is as follows:

Mosher recommended a liberal T-shaped incision which, after
wide elevation and retraction of the flaps, gives a wide shallow field
with maximum visibility. The cross-bar of the T runs parallel with
the lower border of the jaw and about one-half inch below it. The
vertical part runs downward just anterior to the anterior margin of
the sternomastoid muscle. The incision goes through skin, subcu-
taneous fat, superficial fascia, and platysma muscle down to the
superficial layer of the deep fascia which covers the salivary glands.
There is often a great deal of edematous swelling of these tissues.
After exposure of the submaxillary gland the first structure frequently
encountered is the facial, or external maxillary, vein which usually
runs across the lateral part of the submaxillary gland and superficial
to it. The vein is divided between two ligatures. Now comes the
most important step of the operation, namely, identifying and elevat-
ing the submaxillary gland. Its fascia is sometimes rather dense in the
presence of inflammation. It must be identified and incised just above

the hyoid bone. If the hyoid bone is pushed into prominence with the finger from the opposite side of the neck, determination of the correct level is greatly facilitated. This having been determined, the fascia is incised, whereupon the gland looms into view. The inferior border of the gland is elevated with a blunt instrument or finger, working outward toward the lateral part of the gland. At this location one must avoid injuring the facial, or external maxillary, artery which either enters the gland or courses along beneath it to reach the border of the jaw. When the gland is lifted the artery goes with it. The most difficult part of the operation has now been accomplished. The finger is then inserted under the lateral part of the gland in the direction of the angle of the jaw, and the stylomandibular ligament felt. It is then carried upward toward the styloid process deep to the mastoid tip. When the process is felt the finger passes upward beneath it to the base of the skull. It is then in the pharyngomaxillary fossa. It is practically impossible to make a false passage with the finger for this is the path of least resistance, and one gets the sensation that there is no other place the finger could go. If there is a pharyngeal fistula the finger might enter the pharynx, and this has happened to me in cases in which intrapharyngeal incision was done previously. In such cases there is no advantage in enlarging such a fistula, and care should be exercised to avoid that. An abnormally long styloid process may be somewhat troublesome to pass; a short one is an advantage. Identifying the stylomandibular ligament is not of great importance, except that it gives one the assurance that the finger is close to the inner surface of the mandible and will reach the styloid process. The region at the lower pole of the tonsil, where small abscesses are frequently located, is also reached by this procedure. By coming forward and inward at the proper level the region at the base of the tongue and floor of the mouth may be palpated and explored with the finger. The abscess in the mouth floor is thus reached and evacuated. In such cases there is sometimes a fistula communicating with the mouth cavity. There is no advantage in enlarging such a fistula, as the drainage through the external wound will be adequate. Laterally situated cases of Ludwig's angina are ideal indications for the employment of this approach.

It cannot be too much emphasized that the most important step in this operation is the proper exposure and elevation of the submaxillary gland. In cases of Ludwig's angina in which the tissues are greatly swollen with inflammatory infiltration, recognition and identification of structures and tissue planes are indeed difficult, and one must proceed with courage and caution, but with precision. One must identify the hyoid bone, and penetrate the deep fascia above its level. One must learn to work in these inflamed and swollen
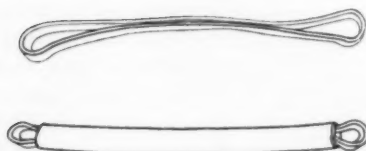
Fig. 2.—A type of rubber drainage material devised to avoid extrusion from wounds in soft parts where movement cannot be entirely eliminated. Tissues protrude into the loop, and hold it in place. It can be made in two sizes: one, a band $\frac{1}{4}$ inch in width by 3 inches in length; the other, $\frac{1}{2}$ inch in width by 4 inches in length. Over it is cuffed a piece of Penrose tubing of such size as to fit snugly, the tubing being cut shorter to allow a loop of band to protrude at each end.

tissues. It is quite different from working on the cadaver where the deep fascia is so close to the skin that it is often incised when the skin incision is made. There is often as much as two to five centimeters of cutaneous and subcutaneous edema, the more so in an obese patient. Cultures are made from the wound and from the pus which is evacuated.

At the conclusion of the operative procedure the drain must be carefully placed in the pharyngomaxillary fossa or wherever the abscess was encountered. As previously stated, I prefer the soft rubber cuffed drain. In cases in which the internal jugular vein has not been exposed for ligation, the vertical part of the incision may be closed with sutures, and perhaps also the ends of the horizontal part. Maintenance of a large incision is not necessary when the drain is accurately placed and held in position. In partial closure of infected wounds I prefer chromic catgut skin sutures because they resist infection. Metal skin clips may be used. In cases in which ligation or resection of the internal jugular vein has also been done, a soft Penrose drain should be placed on the sheath and brought out through the lower end of the vertical incision. Usually the drain in the pharyngomaxillary space is kept in for about six days. On the day before removal it is pulled out about half way; or it may be pulled out a fraction of an inch on each of several days. If local and general symptoms and signs indicate that there is considerable inflammatory swelling and discharge with some sepsis, drainage should be continued until all local and general signs of inflammation and sepsis have disappeared. In cases in which there is no necessity for exposing the carotid sheath the vertical part of the incision may be omitted; and in that event the horizontal incision should be a little longer and placed about midway between the lower border of the jaw and the
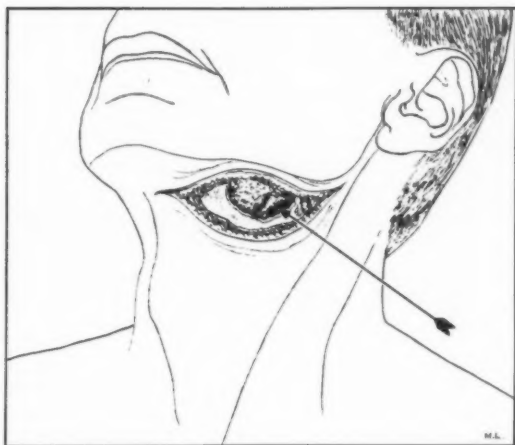
Fig. 3.—Drawing showing surgical exposure of the submaxillary salivary gland. Shaded area (arrow) at the lateral aspect of the gland is the place where blunt instrument or finger is inserted to reach the pharyngomaxillary fossa.

hyoid bone, so that adequate exposure may be obtained by wide elevation and retraction of the flaps.

Approach at the Angle of the Jaw. A rapidly performed and direct approach to the pharyngomaxillary fossa may sometimes be desirable. Such an approach is available by blunt penetration of the deep fascia exactly contiguous to the angle of the jaw. At this point the fascia is tough and dense because it is within the area where the superficial and deep layers of fascia covering the salivary glands come together in union between the submaxillary and parotid salivary glands. If a closed clamp is thrust through and the opening made large enough to permit the introduction of the finger, this may be passed upward to the styloid process and into the pharyngomaxillary fossa. More force is required to carry out this maneuver than is necessary by the submaxillary route. It is important that the finger keep close to the internal pterygoid muscle on the inner side of the mandible as it is advanced.

SUBMAXILLARY SPACE INFECTION

In cases of Ludwig's angina type of infection, whether of dental or non-dental etiology, the submaxillary approach is the approach

of choice. Through this, access to any part of the region above the
mylohyoid muscle may be had.   In these the patency of the airway
must always receive consideration, and if there is any doubt a tra-
cheotomy should be done first.   Trismus is usually pronounced.

SUBMENTAL SPACE INFECTION

When the infection is predominant in the mid-region of the
neck an approach between the muscles of the two sides of the tongue
in the midline may be the most direct.   A horizontal incision is
made through the skin, exposing the deep fascia covering the mylo-
hyoid muscles.   A vertical incision reaching from the symphysis of
the mandible to the hyoid bone is then made in the fascia exactly in
the midline. A closed clamp is then thrust through the median raphe
between the mylohyoid muscles toward the tongue base.   Surprisingly
large quantities of pus are frequently evacuated through this ap-
proach.   It is simple, direct, and no important structures are en-
countered.   Further exploration can then be caried out with the
finger.   The cuffed drain is then inserted, and the skin wound par-
tially closed on each side.   The tongue may be explored throughout
its entire thickness, and laterally to either side between the several
muscle planes. This may be termed the midline suprahyoid approach.
It is simple and direct.   As with all submaxillary region infections
in which trismus and impairment of the airway are common a tra-
cheotomy must often be done first.

PAROTID SPACE INFECTION

The skin incision may be single, but the capsule of the gland
may be opened in several places.   When the infection first involves
the retromandibular, or pharyngeal, process of the gland, from the
parapharyngeal area, it is best dealt with by draining the pharyngo-
maxillary fossa through the submaxillary fossa approach. When the
infection is primary in the parotid gland an incision over the prom-
inence of the parotid swelling parallel to the branches of the facia
nerve may suffice.   A better and more extensive exposure is obtained
by Furstenberg's incision through the skin from close to and above
the tragus to the level of the angle of the jaw and turning forward
to below the angle.   The skin is then elevated from the fascia cover-
ing the gland, and incisions are made through the fascia in a direction
paralleling the branches of the facial nerve.   As many incisions are
made in the fascia as necessary to reach all visible pus accumulations.
Search may be made with a blunt instrument such as an artery clamp.

Bezold's Abscess. This may be drained by incising over its
prominence just below the auricle along the anterior border of the
sternomastoid muscle, and directing the approach toward the digastric

Fig. 4.—Incision for midline suprahyoid approach in cases of the sub-maxillary type of infection (Ludwig's angina), through skin and subcutaneous tissues above hyoid bone.
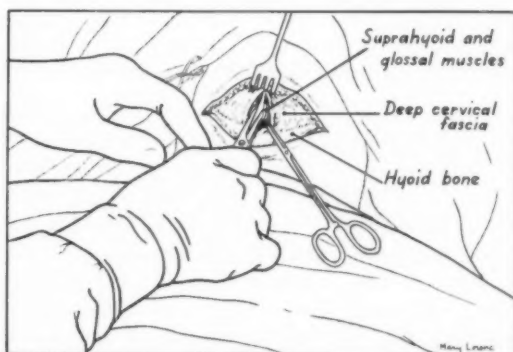
Fig. 5.—Insertion of the closed forceps through the midline incision in the cervical fascia, into the median raphe of the mulohyoid and lingual muscles. This midline incision through the deep fascia extends from symphysis menti to hyoid bone.

groove and root of the styloid process, beneath the sternomastoid muscle. By using the finger from below the parotid gland, deep extension inward may be followed and reached. At the same time a complete simple mastoidectomy should be performed if it has not already been done. By performing the mastoidectomy first, the abscess will be reached when the tip is removed. The incision through the soft parts may then be extended downward and forward to increase the exposure.

VISCERAL FASCIA INFECTION

Incise over the abscess if one has formed. In those in which there has been no localization the incision is made along the anterior border of the sternomastoid muscle, exposing and identifying the sheath. The muscle and the carotid sheath are retracted laterally. Then the larynx, trachea and esophagus are exposed, identified, and retracted medially. The drain is placed along the viscera. Below the level of the larynx another approach may be used. The viscera may be quickly exposed by incising through the isthmus of the thyroid gland from above, ligating it with suture ligatures on each side. Then the gland is gently separated from the trachea, and the recurrent laryngeal nerve and thyroid vessels identified and avoided. A drain may then be placed along the viscera.

POSTERIOR TRIANGLE INFECTION

The most common type of infection in this triangle is abscess of the lymph nodes. When this lies entirely superficial to the deep cervical fascia, and has no deep ramifications it may be dealt with by simple incision over the point of fluctuation, and packed lightly with gauze.

There is a type of infection in the upper part of the occipital triangle characterized by abscess, fairly large, which involves nodes in the uppermost part of this triangle, and which is secondary to inflammation in the pharynx. Frequently there will be inflammatory swelling of the lateral pharyngeal wall behind the posterior pillar indicating infection in the posterior compartment of the pharyngomaxillary space. There are throat symptoms such as sore throat and painful swallowing. In this type of case, when the abscess is opened by incision along the posterior border of the sternomastoid muscle the finger can be passed inward toward the styloid process, keeping close to the paravertebral muscles, and the innermost recesses of the cavity in the parapharyngeal region reached. In bygone years many of these were treated by the general surgeon by this approach.

421 Huguenot St.

REFERENCES

1. Beck, August L.: A Study of Twenty-four Cases of Neck Infection, Trans. of Acad. of Ophthal. & Otolaryng., 1932.

2. Mosher, Harris P.: Deep Cervical Abscess and Thrombosis of the Internal Jugular Vein, Laryngoscope (June) 1920.

3. Mosher, Harris P.: The Submaxillary Fossa Approach to Deep Pus in the Neck, Trans. Amer. Acad. of Ophthal. & Otolaryng. 19-36, 1929.

4. Thomas, T. Turner: Ludwig's Angina, an Anatomical and Statistical Study, Annals of Surg. 47:61-183, 183-312, 312-373 (Feb.) 1908.

5. Mosher, Harris P.: My Milestones, The Laryngoscope 56:639-652 (Nov.) 1946.

6. Welch and Lewis: Antibiotic Therapy, The Arundel Press, Inc., 1951.

7. Blassingame, Charles O.: Angina Ludovici: An Anatomic and Clinical Study, Arch. Otolaryng. 8:159, 1928.

8. Iglauer, Samuel: Surgical Approaches to Deep Suppuration in the Neck and Posterior Mediastinum, Arch. Otolaryng. 21:707-716 (June) 1935.

9. Iglauer, Samuel: Septic Thrombophlebitis of the Neck: Report of a Case with Serious Complications, Arch. Otolaryng. 36:381-384 (Sept.) 1942.

10. Dean, L. W.: The Proper Procedure for External Drainage of Retropharyngeal Abscess Secondary to Caries of the Vertebrae, J. of Laryngol., Rhinol. and Otol. (Sept.) 1918.

11. Orton, H. B.: Deep Infections of the Neck, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 47:61 (Mar.) 1938.

12. Orton, H. B.: Deep Infections of the Neck, J. A. M. A. 120:873-879 (Nov. 21) 1942.

13. Hochfilzer, John J.: Phlegmon of the Neck and Osteomyelitis of the Mandible Following Tonsillectomy, Arch. Otolaryng. 12:175 (Aug.) 1930.

14. Hauser, Carl Musser: Ludwig's Angina: Intraoral Incision in Infections of the Floor of the Mouth, Arch. Otolaryng. 16:317 (Sept.) 1932.

15. Ashhurst, A. P. C.: Ludwig's Angina, Arch. Surg. 18:2047 (May) 1929.

16. Richards, Lyman: Peritracheal Abscess, Arch. Otolaryng. 11:336, 1930.

17. Richards, George L.: Phlegmon Starting as Peritonsillar Abscess and Extending Downward as Far as the Second Ring of the Trachea, Laryngoscope 23:835, 1913.

18. Myerson, M. C.: Anaerobic Retropharyngeal Abscess, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 41:805 (Sept.) 1932.

19. Salinger, Samuel and Pearlman, S. J.: Hemorrhage from Pharyngeal and Peritonsillar Abscess, Arch. Otolaryng. 18:464, 1933.

20. Salinger, Samuel: Hemorrhage from Pharyngeal and Peritonsillar Abscess, Laryngoscope 44:765, 1934.

21. Carmody, Thomas E.: Osteomyelitis of the Mandible, Arch. Otolaryng. (Jan.) 1938.

22. Simpson, J. R.: Pharyngomaxillary Infection (with Report of Cases), Trans. Amer. Laryng., Rhin. and Otol. Soc. 44:329-336, 1938.

23. Hall, Colby: Sepsis Following Pharyngeal Infections, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 48:905 (Dec.) 1939.

24. Hall, Colby: Suprahyoid Neck Infections, Trans. Amer. Laryng., Rhinol. and Otol. Soc. 392-406, 1950.

25. Motley, F. E.: Massive Hemorrhages as an Otolaryngological Complication, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 51:737-755 (Sept.) 1942.

26. Woodruff, G. H.: Massive Pharyngeal Hemorrhages, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 54:202-207 (Mar.) 1945.

27. Grove, W. E.: Abscess of the Pharyngeal Space: Massive Hemorrhage, Recovery. A Case Report, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 51:1053 (Dec.) 1942.

28. McCaskey, C. H.: Ludwig's Angina, Arch. Otolaryng. 36:467-472 (Oct.) 1942.

29. Higbee, David: Acute Thyroiditis in Relation to Deep Infection of the Neck, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 52:620-627, (Sept.) 1943.

30. Furstenberg, A. C. and Yglesias, Louis: Mediastinitis: A Clinical Study with Practical Anatomic Considerations of the Neck and Mediastinum, Arch. Otolaryng. 25:539-554 (May) 1937.

31. Furstenberg, A. C.: Acute Suppurations of the Mouth, Pharynx, and Neck, New Orleans Med. & Surg. J. 94:162-169 (Oct.) 1941.

32. Rosenthal, Abraham: Eight Fatal Anaphylactic Reactions to Penicillin, N. Y. State J. of Med. 1485-1487 (May 15) 1954.

33. Dubos, Rene J.: Biochemical Determinants of Infection, Bulletin of N. Y. Acad. of Med. (Jan.) 1955.

XIII

THE PLASMA CELL THEORY OF ANTIBODY FORMATION

ITS IMPORTANCE IN THE INTERPRETATION OF THE PHYSIOLOGY
OF THE LYMPHOID STRUCTURES OF WALDEYER'S RING

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The plasma cell theory of antibody formation offers a solution to many perplexing problems concerning the functions of the lymphatic tissue of Waldeyer's ring as well as those of lymphatic tissue in general.

THE SITE OF ANTIBODY FORMATION

A number of convincing experiments have demonstrated that lymphatic tissue is the probable source of antibodies. McMaster and his co-workers[25] have shown that the lymph nodes, nearest the site of an intradermal injection of bacteria, form antibodies before they appear in appreciable concentration in the blood, and were able to rule out the possibility that antibodies found in the regional lymph nodes are formed elsewhere in the body.[25, 26] They also observed that antiviral substances neutralizing vaccinia virus appear first, in significant amounts, in the lymph nodes nearest the site of an intradermal injection of the virus.[26]

Although antibodies appear to be formed exclusively within lymphatic tissue, the actual mechanism concerned with their manufacture has remained obscure. Since reticuloendothelial cells, polymorphonuclear leukocytes, lymphocytes and plasma cells are all present in lymphatic tissue, each of these, at various times, has been considered to be the potential source of antibodies.

THEORIES OF ANTIBODY FORMATION

The wide acceptance of the reticuloendothelial cell theory of antibody formation was based on the proposition that cells which ingest and digest bacteria are also most likely to be concerned with the synthesis of antibodies.[10] This theory became untenable when Ehrich and his coworkers[14] found that the intravenous injection of small amounts of staphylococcus vaccine caused marked antibody formation with little response of the reticuloendothelium, while large doses of staphylococcus vaccine produced little antibody and enormous proliferation of the reticuloendothelium. They[12] also found

that the formation of antibody in the regional lymph node, following the subcutaneous injection of antigen, was accompanied by marked proliferation of lymphoid cells, but not of reticuloendothelium. Finally, by means of extraction experiments, they demonstrated antibodies in isolated lymphoid cells in titers as high as 1:6,144,[16] whereas macrophages, as well as granulocytes, never contained demonstrable antibody.[13]

Until quite recently, the evidence that lymphocytes were the cellular sources of antibodies appeared to be conclusive. There is unequivocal evidence that antibodies are elaborated within lymph nodes; that the cellular response during antibody formation is chiefly lymphocytic; and that antibodies are present in lymphoid cells during antibody formation. It has been shown that sufficient irradiation to induce destruction of lymphoid tissue also causes marked depression of antibody formation,[18, 27] while increased activity of lymphoid tissue, brought about by the application of dry heat, results in increased antibody formation.[27] Hyperplasia of lymphoid tissue, preceding or accompanying antibody formation, can increase the weight of a lymph node five-fold and, during this time, there may be a rise in the output of lymphocytes in the efferent lymph from 15,000 to 20,000 per cu mm to 60,000 to 100,000 per cu mm, and without a significant response on the part of the reticuloendothelium.[14] However, this mass of evidence, all of which is basically correct, fails to prove that lymphocytes are the cellular sources of antibodies.

The plasma cell theory of antibody formation, first suggested in 1913,[19] attracted little attention until Bing and Plum,[2] in 1937, pointed out that patients with hyperglobinemia (increased gamma globulin content of the blood) also show an increase of plasma cells in the body tissues. Clinically, the highest globulin levels were found in patients with multiple myeloma (plasma cell tumor), whereas in lymphatic leukema (lymphocyte tumor), hyperglobinemia was absent.[9] Later, experimental observations in rabbits[3, 8] demonstrated that intravenous hyperimmunization is associated with marked plasma cell proliferation in the spleen and other organs. The development of techniques for extracting antibodies from plasma cells[4] led to the further study of cellular changes in hyperimmunized rabbits and the finding of an almost pure and often massive plasma cell infiltration of the adipose tissue in the renal sinus; extracts of the infiltrations contained high antibody concentrations but the thymus, which contained a much higher proportion of lymphocytes than plasma cells, possessed very little antibody.[4, 17]

The demonstration that antibodies were formed in tissue cultures led to further investigations.[15] Thus, cultures of splenic tissue from

immunized rabbits revealed that the red pulp, containing abundant plasma cells, formed greater amounts of antibodies than the Malpighian bodies, composed chiefly of lymphoid cells.[15,23] It has also been shown that some cells of antibody forming lymph nodes agglutinate *in vitro* the bacteria with which the animals are immunized,[11] and these agglutinating cells belong to the plasma cell series, whereas typical, large or small lymphocytes or other leukocytes fail to show this phenomenon.

It is significant that the rise in antibodies in the regional lymph node, following the subcutaneous injection of antigen, is associated with the proliferation and maturation of plasma cells in the medullary cords, and accompanied by a definite increase in pentose nucleic acid, most of which is contained in the plasma cells.[11] Of equal importance was the observation that efferent lymph from an antibody forming lymph node may contain as much as four times as many plasma cells as the control lymph.[9]

Ehrich[9] has pointed out that the plasma cell rather than the lymphocyte appears to be the cell producing antibodies solely on the basis of the morphologic appearances of the two cells. The plasma cell has abundant cytoplasm, rich in pentose nucleic acid, while the small lymphocyte has very little cytoplasm and a low concentration of pentose nucleic acid. Moreover, in pathologic conditions, the plasma cell frequently shows retention of protein within its cytoplasm (Russel bodies), while the lymphocyte has never been observed to contain such inclusions.

Since the report of the first case of agammaglobinemia in 1952,[6] additional cases have been reported[7, 20, 22, 35] with findings which further support the validity of the plasma cell theory of antibody formation. Originally believed to be sex linked and congenital, the first case reports concerned children with recurrent infections since infancy, the most common being pyoderma, purulent conjunctivitis, otitis media, purulent sinusitis, pneumonia, meningitis and purulent arthritis. These patients were unable to form antibodies to bacteria and to viruses,[22] and although the infections responded promptly to antibiotic therapy, recurrences soon followed withdrawal of the drugs. Low gamma globulin content was present in the connective tissue obtained by biopsy; at autopsy, there was marked diminution of gamma globulin in the lymphoid tissue.

Later, agammaglobinemia was observed to occur in adults in whom there was no evidence of hereditary or congenital origin.[35] It is now evident that there are two types of agammaglobinemia, one idiopathic and probably congenital, the other acquired or secondary to a disease of the lymphatic or reticuloendothelial systems.

It is of interest to note that secondary agammaglobinemia has been reported in patients with chronic lymphatic leukemia,[5] malignant lymphoma,[1] multiple myeloma[34] and sarcoidosis.[35]

Agammaglobinemia is characterized by normal protein fractionations with the exception of gamma globulin which may be absent entirely or present in such small amounts that it can be demonstrated only by immunochemical methods. The patients with this disease are unable to produce humoral antibodies. In one patient,[35] the tissues of the liver, spleen and lymph nodes showed complete absence of plasma cells.

PRESENT CONCEPTS CONCERNING
THE PLASMA CELL THEORY

Ehrich[9] has summarized the evidence regarding cellular activity in immune body formation. In general, the role of the various cells is as follows:

1. In antibody formation, the role of the reticuloendothelial cells and polymorphonuclear leukocytes is limited to: a) the splitting of aggregated or corpuscular antigen into chemically active antigen molecules; and b) the storage of antigen.

There is incontrovertible evidence that soluble antigen is liberated from corpuscular antigen and transported to the regional lymph node, and that storage of antigen in the reticuloendothelium occurs for as long as 75 days under certain conditions.

Both polymorphonuclear leukocytes and reticuloendothelial cells possess enzyme systems which are capable of degradation but neither type of cell contains sufficient pentose nucleic acid for the manufacture of protein for export.

2. The role of the lymphocyte in antibody formation appears to be: a) the provision of building stones for the synthesis of nucleic acids and proteins; b) to act as a source of materials which stimulate phosphorylation and create energy for the synthesis occurring in plasma cells; and c) to store a portion of the antibodies and gamma globulins formed by the plasma cells.

The degradation of lymphocytes, effected by certain enzyme systems, proceeds largely within lymphoid tissue.

3. The role of the plasma cell in antibody formation is limited to the synthesis of gamma globulin which contains the majority of humoral immune bodies.

It is probable that molecular or corpuscular antigen, such as the unaltered bacterial cell, fails to stimulate the synthesis of antibodies

by the plasma cell. Aggregated or corpuscular antigen must be split by the reticuloendothelial cells and polymorphonuclear leukocytes into chemically active, antigenic molecules before the plasma cell is stimulated to synthesize antibodies. The energy and raw materials, essential for such synthesis, are derived from the products resulting from the degradation of lymphocytes in lymphoid tissue.

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THE ROLE OF THE PLASMA CELL IN THE
LYMPHOID TISSUE OF WALDEYER'S RING

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Waldeyer's ring, consisting of a defensive mass of lymphoid tissue surrounding the entry to the lower respiratory tract and digestive tract, is situated where there is urgent need of a mechanism capable of removing and attenuating microorganisms and developing immunity against them. In the oral cavity and oropharynx, microorganisms in food have a transient contact and, normally, the secretions of the gastrointestinal tract are capable of rendering most of these innocuous. Bacteria, reaching the digestive tract in secretions from the oral and nasal cavities, are similarly altered, inhibited or digested. That immune body formation may also be initiated in the gastrointestinal tract is apparent from the presence of such lymphoid structures as Peyer's patches.

The main responsibility of the lymphoid tissue of Waldeyer's ring appears to be the initiation of reactions which result in the production of antibodies specific for the microorganisms present in the upper respiratory tract and oral cavity. Not only must immunity be established against those microorganisms constantly present in the environment, but also against those arriving, anew or intermittently, in sufficient numbers to alter the flora of these areas. Frequently, survival may depend upon the integrity of this mechanism.

Certain components of Waldeyer's ring, such as the faucial and pharyngeal tonsils, are actually exteriorized lymphatic tissue and, by virtue of their exposed positions, promptly influenced by various local changes. It may be well to emphasize again, however, that the functions of the various units of Waldeyer's ring differ in no important respects from those of the general lymphatic system.[33]

Other factors also facilitate prompt response to changing conditions. In many areas within the crypts, the lymphoid tissue of the tonsils lies unprotected by epithelium, and the curious epithelial structure referred to as lymphoepithelium[21] exhibits prompt responses to changes in the character of the secretions in contact with it. The plasma cell, however, is a fixed cell and does not appear to invade the epithelium.

In contrast to the lymph supply of lymph nodes, in part if not largely derived from the afferent lymph vessels, the lymph supply of the lymphoid tissue of Waldeyer's ring is derived partly from the superficial lymph spaces immediately beneath the basement membrane of the epithelial layer, and partly from the capillary walls of vessels within the lymphoid tissue. The rate of the release of lymph from vessels within the lymph tissue depends upon factors which have been described.[24]

In spite of the absence of afferent lymph vessels, therefore, the lymphoid tissue of Waldeyer's ring obtains an adequate supply of lymph for physiologic purposes and including the synthesis of antibodies. Moreover, the arrangement of the lymph spaces, immediately beneath the basement membrane of the mucous membrane of the pharynx and fauces, is unusually profuse and especially abundant over the various lymphoid masses.[31, 32] The superficial lymph spaces are arranged in a complex, intercommunicating pattern. These posessess no valves but the lymph channels, beneath the superficial or submucosal lymphatics, possess valves. The lymph vessels with valves may serve to control the quantity of lymph in the superficial channels. In addition, lymph vessels in general are susceptible to a vasoconstrictor action capable of blocking the lymph circulation and this action depends directly on the sympathetic stimulation; the lymphangiospastic impulses follow the same course as do the periarterial sympathetic fibers.[30]

The movements of the muscles of the pharynx and fauces during deglutition, respiration and phonation induce extensive to and fro movements of lymph in the superficial, valveless lymph spaces. These excursions of lymph serve to alert the superficial lymphoid structures, such as the solitary follicles, of changing conditions beyond their immediate limits and facilitate earlier immunologic response.

The corpuscular antigen of most bacteria fails to stimulate antibody formation. It is only after reticuloendothelial cells and polymorphonuclear leukocytes split the aggregated or corpuscular antigen of the bacterial cell into chemically active antigenic molecules that antibody synthesis is initiated. Ingestion and digestion of bacteria by polymorphonuclear leukocytes actually begin on the mucosal surfaces and continue as the tissue is invaded. Active antigen is released, therefore, on the surface, in the epithelial layer and in the tunica propria. Further ingestion, digestion and release of active antigen occur when reticuloendothelial cells are reached. Once antigenic materials are made available to the lymph, either in the lymph spaces immediately beneath the basement membrane or elsewhere, transportation to lymphatic tissue follows. Additional antigen is

provided by the activity of reticuloendothelial cells within lymphatic tissue.

Until recently, the presence of reticuloendothelial cells and plasma cells in the lymphoid tissue of the upper respiratory tract, as well as in lymphoid tissue in general, has excited surprisingly little interest. Since the number and the characteristics of the reticuloendothelial cells and plasma cells within lymphoid tissue are closely correlated with the activity as well as the inactivity of antibody formation, information of considerable value may be gained by careful histologic examination of the tonsils and other lymphoid tissue in Waldeyer's ring. As far as the human tonsil is concerned, many histologic observations of the past serve to support the theory that plasma cells in lymphoid tissue are concerned, at least in part, with antibody formation.

The physiologic variations in the mass of the lymphoid tissue of Waldeyer's ring appear to conform with immunologic requirements. The prenatal tonsil alone conforms to the usual concepts of the normal histologic structure of this organ. Inmmediately after birth, the tonsil is contaminated with secretions from the birth canal and with microorganisms arriving in air and food. Thereafter, the size and histologic characteristics of the tonsil depend upon the type and frequency of infections to which it is subjected. Histologically, the postnatal tonsil appears to constantly show cellular evidences of irritation and inflammation.

While the human tonsil, at birth, is so insignificant that it is identified with difficulty, it begins to increase in size at once. This apparent growth is due to the infiltration and proliferation of lymphocytes and the arrival of large numbers of plasma cells. It is likely that the increase in cellular constituents is due to the irritative action of infectious materials reaching the tonsil by way of food and air, because these immediate cellular changes are absent in animals born and reared under aseptic conditions.

The increase in size of the human tonsil, during the first three months of postnatal life, is due almost entrely to the appearance of large numbers of plasma cells. At three months, secondary nodules appear and, thereafter, the proliferation of lymphocytes becomes greatly accelerated. While reticuloendothelial cells are present, their numerical proportion has not been established.

Serum gamma globulin levels are normal in the newborn but drop by about one-third in the first month of life;[33] they remain at low levels for about three months after which they gradually begin to rise and reach near-normal, adult values at the age of two years.[26]

During this early period of the infant's life, infectious diseases are a constant menace, but therapy with pooled human gamma globulin appears to offer a measure of protection.[24] In the light of the plasma cell theory, the early preponderance of plasma cells and the sequence of increasing availability of lymphocytes to furnish the materials and energy for antibody formation appear significant.

The peak in tonsillar size occurs during infancy and early childhood when the incidence of respiratory infections is highest. After six years of age, there is a gradual reduction in the incidence of respiratory infections and the size of the tonsil tends to remain stable. Between the tenth year and puberty, there is a recession in size which has, heretofore, been ascribed to endocrine factors but which may be due in part to a reduction in the immunological requirements. It cannot be denied that the attainment of normal, adult values of serum gamma globulin, just prior to puberty, coincides with beginning involution of the lymphoid structures in Waldeyer's ring. After puberty, the lymphocyte content of these structures progressively decreases while there is disproportional diminution in the numbers of reticuloendothelial and plasma cells. In some pathologic states, the adult human tonsil is capable of overcoming the trend toward involution and atrophy and may reassume the task of forming antibodies.

In old age, the physiologic activity of Waldeyer's ring is at low ebb because the acquisition of immunity to a multiplicity of infectious agents has minimized the need for antibody formation.

Antibodies, specific for some strains of streptococci, repeatedly isolated from cultures obtained from the fauces and pharynx prior to tonsillectomy, have been demonstrated in the excised tonsillar tissue. This tissue also shows proliferation and maturation of plasma cells and a definite increase in the concentration of pentose nucleic acid. If further investigations demonstrate that most of the pentose nucleic acid is contained in the plasma cells of such excised tonsillar tissue, material support will be obtained for the plasma cell theory of antibody formation as well as for the concept that antibody formation in the lymphoid tissue of Waldeyer's ring is accomplished by the mechanism employed by the other lymphatic structures of the body.

SUMMARY

The plasma cell appears to be the only cellular element in lymphoid tissue capable of synthesizing antibodies and gamma globulin. Lymphoid tissue is the site of antibody formation and the lymphoid tissue of Waldeyer's ring contains all of the cellular constituents

essential for the manufacture of humoral immune bodies. Lymph nodes and other lymphatic tissue appear to be the birthplace and graveyard of lymphocytes as well as the source of antibodies. The strategic position and structural modifications of the lymphoid tissues of Waldeyer's ring suggest that the prime function of this tissue is the formation of antibodies and other gamma globulins.

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REFERENCES

1. Arends, T., Coonrad, E. V. and Rundles, R. W.: Serum Proteins in Hodgkin's Disease and Malignant Lymphoma, Am. J. Med. 16:833, 1954.

2. Bing, J. and Plum, P.: Serum Proteins in Leucopenia, Acta Med. Scand. 92:415, 1937.

3. Bjorneboe, M. and Gormsen, H.: Experimental Studies on Role of Plasma Cells as Antibody Producers, Acta Path. et Microbiol. Scand. 20:649, 1943.

4. Bjorneboe, M., Gormsen, H. and Lundquist, F.: Further Experimental Studies on Role of Plasma Cells as Antibody Producers, J. Immunol. 55:121, 1947.

5. Brem, T. H. and Morton, M. E.: Cited by Zinneman et al.[35]

6. Bruton, O. C.: Agammaglobinemia, Pediatrics 9:722, 1952.

7. Bruton, O. C. Apt, L., Gitlin, D. and Janeway, C. A.: Absence of Serum Gamma Globulins, Am. J. Dis. Child. 84:632, 1952.

8. Campbell, B. and Good, R. A.: Antigen-antibody Mechanisms in Neurotrophic Virus Diseases, Ann. Allergy 7:471, 1949.

9. Ehrich, W. E.: Cellular Sources of Antibiodies. Blood Cells and Plasma Proteins: Their State in Nature, Academic Press, Inc., New York, 1953, p. 187.

10. Ehrich, W. E.: The Role of the Lymphocyte in the Circulation of the Lymph, Ann. N. Y. Acad. Sci. 46:823, 1948.

11. Ehrich, W. E., Drabkin, D. L. and Forman, C.: Nucleic Acids and Production of Antibody by Plasma Cells, J. Exp. Med. 90:157, 1949.

12. Ehrich, W. E. and Harris, T. N.: Formation of Antibodies in Popliteal Lymph Node in Rabbits, J. Exp. Med. 76:335, 1942.

13. Ehrich, W. E., Harris, T. N. and Mertens, E.: Absence of Antibody in Macrophages During Maximum Antibody Formation, J. Exp. Med. 83:373, 1946.

14. Ehrich, W. E. and Voight, W.: Uber die Reaktionen des Gefassdegewebs Apparatus auf intravenose Staphylokokkeninjectionen und ihre Bedeutung, Beitr. Path. Anat. u. All. Path. 93:348, 1934.

15. Fagraeus, A.: Antibody Production in Relation to the Development of Plasma Cells, Esselte Aktiebolag, Stockholm, 1948.

18. Hektoen, L. J.: The Influence of the X-ray on the Production of Antiocyte in Antibody Formation, J. Exp. Med. 81:73, 1945.

17. Harris, T. N., Rhoades, J. E. and Stokes, J., Jr.: Study of Role of Thymus and Spleen in Formation of Antibodies in Rabbit, J. Immunol. 58:27, 1948.

18. Hektoen, L. J.: The Influence of the X-ray on theProduction of Antibodies, J. Infect. Dis. 17:415, 1915.

19. Huebschmann, P.: Das Verhalten der Plasmazellen in der Milz bei infectiösen Prozessen, Verhandl. deut. path. Ges. 16:110, 1913.

20. Janeway, C. A., Apt, L. and Gitlin, D.: Agammaglobinemia, Trans. Assoc. Amer. Physicians 66:200, 1953.

21. Kelemen, G.: Histological Test of Tonsillar Activity Based on the Reticuloepithelial Interplay, Arch. Otolaryn. 59:263, 1954.

22. Keidan, S. E., McCarthy, K. and Haworth, J. C.: Fatal Generalized Vaccinia with Failure of Antibody Production and Absence of Serum Gamma Globulin, Arch. Dis. Child. 28:110, 1953.

23. Keuning, F. J. and van der Slikke, L. B.: Role of Immature Plasma Cells, Lymphoblasts and Lymphocytes in Formation of Antibodies as Established in Tissue Culture Experiments, J. Lab. and Clin. Med. 36:167, 1950.

24. Landis, E. M.: Capillary Permeability and the Factors Affecting the Composition of Capillary Filtrate, Ann. N. Y. Acad. Sci. 46:713, 1946.

25. McMaster, P. D. and Hudack, S. S.: Formation of Agglutinins Within Lymph Nodes, J. Exp. Med. 61:783, 1935.

26. McMaster, P. D. and Kidd, J. G.: Development of Antiviral Properties Within Lymph Nodes, Proc. Soc. Exper. Biol. & Med. 34:547, 1936.

27. Murphy, J. B. and Sturm, E.: Comparison of Effects of X-ray and Dry Heat on Antibody Formation, J. Exp. Med. 41:245, 1925.

28. Orlandini, O., Sass-Kortsak, A. and Ebbs, J. H.: Serum Gamma Globulin Levels in Infancy, Am. J. Dis. Child. 84:632, 1952.

29. Reiss, E., Mertens, E. and Ehrich, W. E.: Agglutination of Bacteria by Lymphoid Cells in Vitro, Proc. Soc. Exp. Biol. & Med. 74:732, 1950.

30. Ruszyàk, I.: New Studies on the Physiology and Pathology of the Lymphatic Circulation, Minerva Med. 45:146, 1954.

31. Schenck, H. P.: The Influence of Nasopharyngeal Hyperplasia on the Ear; Histologic Examination of Hyperplastic Lymph Follicles After Irradiation, Laryngoscope 51:780, 1941.

32. Schenck, H. P.: Histopathologic Changes Occurring in Chronic Infections of the Pharynx, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 50:817, 1941.

33. Schenck, H. P.: The Anatomy, Physiology and Pathology of Nasopharyngeal Lymphoid Tissue, Trans. Am. Acad. Ophth. and Otolaryn. 54:479, 1950.

34. Snapper, I., Turner, L. B. and Moscowitz, H. L.: Multiple Myeloma, Grune & Stratton, New York, 1953, p. 83.

35. Zinneman, H. H., Hall, W. H. and Heller, B. I.: Acquired Agammaglobinemia, J. A. M. A. 156:1390. 1954.

XIV

OSTEOMYELITIS OF THE FRONTAL BONE

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The historical background of osteomyelitis of the frontal bone starts with more or less nebulous case histories in antiquity. The medieval period contributed little and the modern period, especially the last three decades, has shown great progress. In spite of this progress the complete answer is not available. The seriousness of the condition as verified by a high mortality rate, has made it a subject of interest to all who practice the art of medicine. The bibliography is so extensive that unless one is willing to devote at least a major portion of his life's work to this subject, all of the available material and its ramifications cannot be covered in detail. There are few graduates of an accepted medical school who have not been subjected to some phases of the available knowledge on the subject. All who have specialized in the field of otorhinolaryngology have been required to obtain at least a superficial knowledge of the subject and some degree of familiarity with its historical background. For your convenience, and as a separate section under the heading "Chronological Outline," certain of the mileposts are presented. This is not necessarily repetitious or dull. Most of you will meet old friends; and those who have not had time to delve into the bibliography will find some points of interest.

ETIOLOGY

The exciting causes of osteomyelitis of the frontal bone are listed as follows:

a.   Trauma—accidental or operative.

b.   Acute sinusitis (frontal) following insult to these structures, particularly as a result of swimming. Acute upper respiratory infections wherein the congestion is sufficient to interfere with drainage from the frontal sinuses. Acute exacerbations from a chronic frontal sinusitis.

c.   Dental infections.

d.   Distant infections—hematogenous origin.

Any of the pyogenic organisms have the ability to produce osteomyelitis. The staphylococcus, streptococcus, and pneumococcus are by far the more common ones.

The mechanism of the production of osteomyelitis of the frontal bone is usually accepted as being due to a pressure necrosis, which is explained on a basis of local pressure anemia. This affords an opportunity for a retrograde thrombophlebitis. A knowledge of normal embryology, histology and gross anatomy of the paranasal sinuses and associated structures is essential in order to understand the pathways of infection. The veins of Breschet, the lakes of Trolard, and the Haversian canals play an important role. The suture lines of the skull bones offer themselves as a poor barrier to the extension of the infection.

The macroscopic appearance of the bone during the early phases presents little if any change from normal. The diploe later become hyperemic and drops of pus may be found scattered throughout this structure. At the end of approximately ten days, the bone becomes soft and at this time the medullary spaces will exude pus. There is considerable granulation tissue and definite thrombosed veins. As the condition progresses, the bone becomes hemorrhagic in appearance, and later quite dark. Paraosseous abscesses occur and in the later stages one finds sequestration of the bone.

The microscopic appearance of the bone during similar periods presents first edema of the myeloid tissue followed by inflammation, resorption of bone, granulation tissue and frank pus. At about this stage one can anticipate some degree of sequestration. Osteoclasts and osteoblasts are a common finding. There is definite evidence of invasion of the Haversian canals.

This is a brief list of the complications of osteomyelitis of the frontal bone: (a) extra-dural abscess, (b) meningitis, (c) thrombophlebitis, (d) cerebral abscess, (e) pachymeningitis, (f) meningoencephalitis, (g) intradural abscess.

The two main types of osteomyelitis of the frontal bone are: (a) The acute fulminating type. (b) The chronic type, typified by simple perforation and localized pathology.

The mortality rate of osteomyelitis prior to the employment of adequate antimicrobial therapy was high. It was placed at one hundred per cent by McKenzie in 1913, and similar figures were presented as late as 1929 by Campbell. The fact that the latter was an advocate of the intranasal approach and routinely used the rasp may have had some bearing on his results. Bulson (1925) reported on

55 cases. Twenty-nine postoperative, 79 per cent mortality; twenty-six spontaneous, 48 per cent mortality.

The present day widespread and more or less intelligent use of the antimicrobials, either singly or in combination, has definitely influenced the incidence of osteomyelitis of the frontal bone. In addition, they have been primarily responsible for the reduction of the thirty per cent mortality rate, which was the best that could be obtained by extensive and adequate surgery alone to the present day 3.0-4.0 per cent. This reduction in mortality rate was accomplished by the ability of the antimicrobials to completely control the very early cases wherein there is an adequate blood supply. In the established cases they assist the defense mechanisms of the body to stop the spread of the infection by establishing satisfactory barriers. The possibilities for a properly selected antimicrobial to exert its beneficial effect are entirely contingent on the presence of an available blood supply. If the condition has progressed to a point of necrosis and abscess formation, surgical drainage is not only indicated, but is essential for complete recovery. This fact has been proved and there are few of the ultraconservatives who still persist in holding out for the continuation of medical therapy as a substitute for surgical drainage. The proof of this contested point was obtained only by the expenditure of much time, thought and energy on the part of the surgeon, and at a rather high cost, both physical and financial, to the patient.

The intelligent use of the antimicrobials has permitted considerable change in the surgical techniques employed to remove necrotic bone and evacuate frank pus. It is no longer necessary to remove 2.0 cm to 2.5 cm of healthy bone surrounding the osteomyelitic area in order to have relative assurance that the margins of the process have been encompassed and removed. This naturally results in less deformity, and in many instances, obviates the necessity for secondary plastic procedures. It is now possible to confine the surgical removal of bone to that which is macroscopically involved. The antimicrobials assist the natural defense mechanisms to control the microscopic infection in the surrounding normal bone where there is an adequate blood supply. This affords the additional advantage of permitting primary closure. In many instances a coronal hairline incision may be, or even should be employed instead of the more disfiguring inverted "T" incision. The management of the flap as an entire unit maintaining the blood supply of the periosteum, its proper care during the surgical procedure, and the accurate closure in layers maintains an excellent blood supply for the periosteum. The wounds

heal rapidly, and the periosteum does marvelous things in the way of bridging gaps and laying down new bone when its blood supply is intact.

In accomplishing the ablative procedure, the coronal incision usually does not afford sufficient exposure to permit adequate removal of the ethmoid cells. If an ethmoidectomy is required for the removal of the infected cells, which is almost always the case in the the chronic conditions, the classical external ethmoidectomy is done through a separate incision. It is definitely advantageous to retain as much of the good bone of the supraorbital ridges as is possible. It is therefore better to have two inconspicuous incisions and little or no structural deformity than it is to produce marked deformity in the hope of avoiding an additional scar. Other than these technical changes, the fundamental surgical principals must be followed as meticulously as was the case prior to the use of the antimicrobials. In spite of the temptation to depend on the antimicrobials to clean up neighboring infection, it is still imperative that in all cases, no matter how normal the inner table bone may appear, representative sections of the inner tables of both frontal sinuses and all of that of the grossly osteomyelitic frontal bone be removed. This allows direct inspection of the dura. It is only in this way that epidural abscesses will be found.

<div align="center">COMMENT</div>

Osteomyelitis of the frontal bone without involvement of the frontal sinus does occur in a very small per cent of cases. By far the majority of the cases have their inception in pathology in the mucous membrane of the frontal sinuses. Such being the case, it is impossible to divorce the pathology in the two areas and treat either as a single entity. Attempts to do so have been universally disastrous. The proper care of the initial stage of infection in the frontal sinus will forestall the spread of the infection into the surrounding bone. This statement would hold true for the acute fulminating type if it were not for the fact that the time element between initial infection in the mucous membrane of the frontal sinus and the spread to the surrounding bone, by an osteothrombophlebitic process, is so short that, by the time attention is drawn to the fact that there is any pathology present, it is already out of the confines of the frontal sinus.

Thus far the advantages of the intelligent use of the antimicrobials has been stressed. The disadvantages of incorrect and inadequate therapy warrant some mention. Perhaps it is because they are rare that the cases of ineffectual and inadequate therapy stand out in one's mind. There are instances where patients were given two

"shots of penicillin," had marked regression of all of the unpleasant symptoms associated with the acute phase, and after ten or more days of apparently normal existence woke up one morning with a swollen forehead and a severe headache. These patients are rightfully vindictive. This recurrence of the original infection by a rejuvenated bacterial flora may sometimes be controlled by a massive and prolonged treatment with the original antimicrobial. Since penicillin is the usual drug used by individuals who feel pleased with the temporary results obtained from one or two injections of this drug, and therefore discontinue further therapy, subsequent use of penicillin may prove to be ineffective due to the fact that the organisms have become resistant. At this stage it is often difficult to obtain suitable material for culture and isolation of the infecting organism. Therefore it is impossible to run sensitivity tests. If suitable material for culture and isolation of the organisms is not available, and if further antimicrobial therapy is selected as the treatment of choice, at least on a trial basis, some one of the combinations of drugs, even though more toxic, should be employed. If the response is satisfactory, and the x-rays show no evident bone destruction, the therapy should be continued for a period of at least two weeks. When there is roentgenologic evidence of osteomyelitis, surgical intervention is imperative. It was particularly interesting to follow one virile patient through four "medical cures" during a period of five months before both the patient and the medical adviser could be convinced that surgery was necessary. The fact that the last exacerbation was accompanied by a convulsive seizure, followed by several hours of unconsciousness, may have played some role in settling the long-standing argument. In this instance, surgery demonstrated a diffuse, spotty osteomyelitis and the presence of an extensive epidural abscess.

The excellent surgical results obtained in another individual who had received inadequate treatment, and who during the course of his medical trial showed evidence of intracranial invasion, which was found to be a large frontal lobe abscess, are somewhat dimmed by the fact that the patient now has epileptiform seizures. The recovery from the surgery was prompt and satisfactory. The deformity was minimal, and there was no further spread of infection. For the succeeding four years the patient was symptom free and led a normal life. The initial seizure some four years after the surgery followed a mild drinking bout. The seizures were infrequent at first, and of only moderate severity. They incresed in number and severity and have been controlled only after considerable therapeutic experimentation.

The injudicious use of combinations of the antimicrobials is stupid therapy, even though there are instances wherein the result was little

short of a miracle. Too often the combinations of drugs are pre-
scribed with little real knowledge of the active ingredients, and with
great amounts of hope. Hope that the drug will work. Hope that
the patient will get well. Hope that there will be no complications,
and after tangible reasons for hope have been used up, just plain
hope. It is suggested that each newly proposed combination of anti-
microbials be scrutinized carefully as to the actual constituents, and
as to the therapeutic action of the drugs on the basis of their individual
qualities, additive values and synergistic properties as determined in
the laboratory, and by clinical trial. This knowledge is essential in
order to intelligently evaluate the claims made in the brochure put
out by the particular pharmaceutical company sponsoring the particu-
lar combination of antimicrobials. The stated claims made by the
producer are factual and truthful, but may be confusing, or even
misleading, if you are not fully familiar with the available knowledge
on the subject, terminology, and methods employed to obtain the
stated results.

CONCLUSIONS

1. Osteomyelitis of the frontal bone is still a clinical entity.
In spite of modern advances it is accompanied by a reduced but still
formidable mortality rate.

2. The intelligent use of the antimicrobials is of great assistance
in controlling or eliminating the early phases of the infection, and has
proved invaluable in assisting to confine the infection within the
frontal bone.

3. The ability to limit the spread of the infection and the rela-
tive assurance of maintaining the infection within these bounds per-
mits less radical surgery.

4. The majority of cases of osteomyelitis of the frontal bone
have their origin in the frontal sinuses. It is therefore impossible to
separate these two conditions and treat either one as a separate entity.

5. No surgical procedure for osteomyelitis of the frontal bone
should be considered adequate unless the dura over both frontal lobes
has been exposed sufficiently to assure the absence of an epidural
abscess.

6. The antimicrobials have permitted several changes in sur-
gical technique, particularly as regards type of incision, the amount
of bone that must be removed, and primary closure of the skin flaps.

7. There is no antimicrobial or combination of antimicrobials
which has been found to be a substitute for surgical drainage.

8.   The beneficial effects of the antimicrobials cannot be obtained unless there is an adequate and functioning blood supply.   An abscess cavity filled with purulent material has no blood supply.

9.   A chronological outline of some of the mileposts in the historical background of osteomyelitis of the frontal bone is presented in the hope that it will refresh your memory, and perhaps stimulate new interest in this subject.

CHRONOLOGICAL OUTLINE

1760   Percival Potts—puffy tumors.

1859   Chassignac—suspected that pericranial and extradural abscesses are often associated with osteomyelitis of the intervening bone.

1879   Lannelongue and von Bergman—independently recognized that Chassignac's suspicions were a fact.

1882   Fischer—"apparently spontaneous inflammation of the skull is probably caused by progressive inflammation of the neighboring tissues."

1884   Ogston—First to report the opening of the frontal sinuses for drainage of infection.

1894   Jansen—cardinal principles of operative procedure.

1898   Riedel—First described the complete obliterative operation on the frontal sinus.

1899   Luc, of Paris and Tilly, of London—each presented a case of osteomyelitis secondary to accessory nasal sinus disease.   These are the first recorded cases where the sinuses were recognized as being the etiologic factor.

1903   Killian—improved operative technique.   Removed the entire floor and the external wall of the frontal sinus.

1904   Schilling—comprehensive study of the micro- and macroscopic changes. He laid the groundwork for the present knowledge of the pathogenesis and pathologic picture.

1905   Ingalls—suggested a gold tube to keep the nasofrontal duct patent.

1905   Logan Turner—presented two principles:
    1.   Sinus preserved.
    2.   Sinus obliterated.

1908   Knapp—first to approach the sinus externally through the floor.   If the sinus was large, he made an opening through the anterior wall in order to reach the upper part of the sinus.   This approach was later (1921) popularized by Lynch.

1910   Watson Williams—first to mention postoperative cicatricial stenosis of the nasofrontal duct.

1913   Dan McKenzie—a monumental contribution to the management, care, and
and   surgical approach to osteomyelitis of the frontal bone.
1927

1917 Lothrop—indicated the necessity for exploring both frontal sinuses and the importance of having a large opening in the nose. Among other things, he advocated the use of the rasp in the nasofrontal duct.

Tilley—presented the first case of initial external drainage and three weeks later extensive removal of bone and obliteration of the frontal sinus. The case was not one of the extreme fulminating type. Tilley is a precursor for J. M. Brown's teaching.

1921 Lynch—popularized the operation now known under his name. He was antedated by Knapp and Jansen.

1926 Sewall—local anesthesia—less bleeding.

1927 Lillie—advocated a two stage operation.

1928 Mithoefer—first to advocate grafts to line the nasofrontal duct.

1930 Cullom—considered Riedel's operation too deforming and the Killian operation as ineffectual. He advised intranasal work first which should be followed by a Lynch operation.

1931 Furstenberg—adequate surgery. Sound principles.

1933 Ferris Smith—advocated ethmoid, frontal operations with grafts.

Hilding—experimental animals (dog). If extensive strips of mucous membrane were removed, scar tissue bands formed and these interfered with the mucus flow. Most animals had scattered cysts, (similar to the infected pockets so frequently described at secondary and tertiary operations on the human).

Mosher and Judd—definite contributions as to the surgical handling of osteomyelitis of the frontal bone.

1934 Sewall—a new type of mucosal flap.

1936 Diggle and Cawthorne (England)—advocated extremely conservative treatment.

1936 Reaves—demonstrated the practical application of Mosher's dictum in regard to the nasofrontal duct.

1937 Adson and Hempstead—used an inverted "U" incision. Recommended leaving the external table but take down the posterior wall to allow the brain and dura to approach the anterior wall. Used tincture of iodine in an attempt to destroy the sinus mucous membrane.

1939 Patterson—first to suggest chemotherapy (prontosil).

1940 Kettel—coronal incision.

Van Alyea—stated that the radical removal of the sinus mucous membrane was seldom indicated.

1942 Fred—predicted that the sulfonamides would convert the fulminating types into the slowly progressive or localizing types, thus permitting more conservative treatment.

1944 Iglauer, Kirby and Hepp, Putney, Colbert—four articles on the use of penicillin.

1945 Schnitker and McCarthy—emphasized that penicillin is not a substitute for surgery.

Goodale—tantalum tube to keep the nasofrontal duct open.

1947   Negus—(expression of prevalent trends in England at that time) advocated a small incision, did not remove the floor of the sinus, did not curette out the mucous membrane, but did enlarge the nasofrontal duct.

   Erich and New—recommended the use of an acrylic obturator instead of either the gold tube of Ingalls or the tantalum foil of Goodale.

1952   Boyden—a modification and extension of the works of Mithoefer, 1927, and Sewall, 1934.

PLASTIC REPAIR

1938   O'Connor—preserved isocartilage grafts.

1940   New and Dix—autocartilage grafts.
   Kazajian—used bone, cartilage, vitallium, tantalum and the acrylics.

1943   Peer—diced cartilage.

1949   Lierle and Huffman—diced or small pieces of autogenous cancellous bone.

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XV

UNDERGRADUATE AND GRADUATE TEACHING IN OTOLARYNGOLOGY

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One of the indirect benefits of this antibiotic era and its impact on the specialty of otolaryngology is the inquiry that has been directed into the teaching of otolaryngology to the undergraduate, and the adequacy of a resident's training in a particular institution approved for this training. This inquiry was stimulated a few years ago when it became apparent that the number seeking residency training in otolaryngology was diminishing and statements were made by some that our specialty was a dying one.

There then began a process of self-examination. The American Board of Otolaryngology established within its organization committees to study undergraduate and graduate teaching; the Teachers' Section of the American Academy of Ophthalmology and Otolaryngology was revived; and the American Medical Association, the American College of Surgeons and the Board combined their appraising facilities for reviewing residency programs and revising the approved list.

It was generally recognized that the character and scope of our activities had changed in some aspects of our specialty. A number voiced an optimistic outlook toward our future but a few were pessimistic.[1-5, 7-11]

An editorial in the *Journal of the American Medical Association*[6] suggested the need for our self-appraisal in a statement to the effect, "That only in a climate of continuously free and open discussion can a valid, objective assessment of the future of otolaryngology be made. Otolaryngologists have much to gain by threshing out the problem calmly and deliberately rather than by ignoring it."

In 1950, the Teachers' Section of the American Academy of Ophthalmology and Otolaryngology[12] was revived. The earlier history of this Section will be commented on later. In that year undergraduate instruction in otolaryngology was examined by a study of a cross-section of such instruction as offered in the 80 medical schools of the United States and Canada. This brought out the wide dis-

parity in the number of hours allotted to otolaryngology in the undergraduate curricula.   An obvious conclusion was that a department of otolaryngology is only as strong as its leadership and where there is strong leadership the undergraduate is exposed to a picture of otolaryngology that gives him an adequate conception of the possibilities of the specialty.

At that meeting requests were made that this Teachers' Section formulate minimum requirements for an adequate program for undergraduate instruction and to send a copy of these requirements to the deans and heads of departments in all the medical schools.   It was agreed that these minimum requirements should be reviewed at the 1952 meeting of the Teachers' Section.   This was done.   As finally approved the recommendations were identical with those presented at the 1949 meeting of the Academy by Dr. LeRoy Schall, chairman of the Committee on Undergraduate Teaching of the American Board of Otolaryngology.   The following communication was mailed to all deans and heads of departments:

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

RECOMMENDATIONS OF THE

TEACHERS' SECTION — COMMITTEE FOR

OTOLARYNGOLOGY

REGARDING

UNDERGRADUATE TEACHING IN OTOLARYNGOLOGY

Surveys have indicated that there is a great variation in the amount of instruction in Otolaryngology offered undergraduate students in the 80 medical schools of the United States and Canada, and that in many of these schools the program of instruction is obviously inadequate.   The importance of a good program of undergraduate teaching is indicated by some surveys that have shown that approximately 25 per cent of the activity of the general practitioner in rural communities is concerned with disorders in the ear, nose or throat.

The Committee on Undergraduate Teaching of the American Board of Otolaryngology and the Teachers' Section (Otolaryngology) of the American Academy of Ophthalmology and Otolaryngology make the following recommendations regarding minimum requirements for an adequate program of undergraduate teaching in Otolaryngology:

1. Anatomy of the ear, nose and throat in the fresh-
    man year by an otolaryngologist.

2. Two hours of orientation and four hours of phys-
    ical diagnosis in the sophomore year.

3. Forty hours of didactic and clinical instruction,
    preferably in the junior year—arranged accord-
    ing to the facilities available and discretion of
    the head of the department of otolaryngology.

4. Elective courses in the senior year.

A Division of Otolaryngology is often under a Department of
Surgery. Usually, under this arrangement, the teaching of the spe-
cialty suffers. Since today the practice of Otolaryngology is as much
medical as it is surgical, it is the recommendation of the above groups
that A DEPARTMENT OF OTOLARYNGOLOGY SHOULD BE AUTONOMOUS.

August 1, 1952

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It was not anticipated that these recommendations would pro-
duce any startling changes in American undergraduate otolaryngol-
ogy. Improvement on a local level would obviously require initi-
ative on the part of the head of otolaryngology in the particular
institution concerned. However, it was expected that these recom-
mendations would bolster the requests made by a head of a depart-
ment to his dean or to a curriculum committee in his effort to build
up an adequate program of undergraduate teaching.

At the 1951 meeting of the Teachers' Section[13] this self-appraisal
continued under the title: "Have the Opportunities in Otolaryngol-
ogy Decreased or Increased in the Past 15 Years?" Dr. Howard
House spoke for Otology, Dr. Jerome Hilger for Rhinology and
Dr. Howard McCart for Laryngology. Each took stock of the many
problems related to his segment of the specialty of otolaryngology
and the challenge of these problems. The listener must have left

that meeting with the feeling that the scope of otolaryngology in the present day is very broad, presents many challenges in its unsolved problems, and offers medical and surgical opportunities so numerous that it is difficult for one man's work to include the complete specialty in his activities.

A natural question followed. What are the opportunities to get adequate training in all phases of this specialty? In commenting on this Dr. Dean Lierle from his observations as Executive-Secretary of the Board pointed out the fact that there is a lack of uniformity in the quality of training offered by the various institutions now approved for residency training. Many training centers do not have the material and the staff to train residents in the broad field suggested.

It seems evident that more uniformity in the quality of training will develop as the approval of institutions for residency training is limited to those centers that have adequate material and staff. The applicants for residency training are now seeking first the positions in institutions where there is offered a three year program to include training in the basic sciences and a well-rounded, graded clinical experience with increasing responsibility. This apparently can not be found in a majority of those on the approved roster.

The principal discussion at the meeting of the Teachers' Section[14] in 1952, centered about full time versus part time teaching in otolaryngology. Dr. John Kirchner presented the views of the full time teacher and Dr. Albert C. Furstenberg discussed part time faculty services. Obviously, each makes its contribution.

The ideal situation, other things being equal, would seem to be a so-called geographical full time arrangement in which the head of a department is provided an adequate budget, which includes his basic salary, and then is permitted to care for private patients within the teaching center of which he is a part. This plan is operative in several medical schools. In some instances the fees from private patients' care are used to augment the departmental budget. Under these circumstances the faculty salaries are somewhat higher than would otherwise be the case.

In 10 of our 80 medical schools, there are now what may be classified as full time heads of otolaryngology. In most of these the arrangement is a "geographical" one. Several more schools are on the verge of instituting full time heads. It is probable that this trend will continue with the widened scope of otolaryngology, the need for more comprehensive undergraduate and graduate teaching, and programs for research.

The discussion at the Teachers' Section meeting in 1953[15] again returned to undergraduate teaching.   Dr. Francis L. Lederer critically examined this from the standpoint of our objectives.   He recommended a better correlation between otolaryngology and other fields of medicine and surgery by spacing the teaching of the subject over the entire four years as follows:

First Year
> Correlative teaching in the basic sciences.

Second Year
> Co-operative presentation of techniques of examination in studying growth and development and as part of the instruction in the physical examination of the patient.

Third Year
> Survey of such areas of otolaryngology as maxillofacial surgery, audiology and broncho-esophagology by means of clerkships, audiovisual presentations and outpatient clinics.

Fourth Year
> Comprehensive evaluations of the total person in demonstration clinics, in which student discussion is encouraged on a physician-consultant basis.

Also on the agenda of the 1953 meeting was the revelation of a plan in operation by Dr. Joel Pressman to train "Head and Neck" specialists rather than in their designation as specialists in "Ear, Nose and Throat." His viewpoint was expressed in part as follows:

"Over many generations it has become established, whether by intent or accident I cannot say, that surgery would divide itself into specialties limited by anatomical structures, and there developed in keeping with this the specialties of urology, the surgery of the bones and joints, the eye, the brain, the thorax, ear, nose and throat, and others.   As long as this scheme of things persists and surgery is divided upon anatomical lines, teaching and specialization can be orderly and we can arrive at a logical scheme of things.   But there has come upon the horizon complicating factors such as the development of specialties of tumor surgery and plastic surgery which, though worthy in themselves, destroy the concept of an anatomical approach to specialization.

"We cannot specialize two ways at the same time.   We cannot, on the one hand, create surgical specialties of anatomical divisions of the body and on the other have inroads made upon them by specialists who would, for instance, treat the tumors of the area, or still others who would make further inroads by claiming that the plastic or reconstructive features of surgery fall within a specialized field which is not anatomical.   As for plastic surgery without an element of plastic repair.   This applies whether the surgery is within the confines of a body cavity such as the thorax or abdomen or within the area of the head or neck.

"It is my contention that plastic surgery, tumor surgery of the region, and head and neck surgery are all one and the same.   How can they be logically separated and taught as three specialties?   How can one operate upon a tumor of the neck without being a plastic surgeon, a tumor surgeon, and a head and neck

surgeon all at the same time? How thin can one cut a cake? There must be some limit somewhere. There will never come a time when plastic surgery and surgery of the head and neck can be divorced from each other effectively and taught separately.

"If we were to start all over again and there were no surgical specialties, the ideal would be to create a single specialty of head and neck surgery to include everything above the clavicles with the exception probably of the eye and brain. It is still the ideal, but we are not permitted to start all over again and must accept some sort of compromise, men still being jealous of their own prerogatives, which they only seldom are willing to sacrifice for the common good. We, therefore, need to plan our teaching approach to the problem upon the spirit of compromise. First, we will not succeed at all if we follow a policy of exclusion. To create a well rounded teaching program we need to include teachers in fields other than Otolaryngology rather than exclude them. We will not succeed by trying to exclude anyone with a legitimate right to teach or operate in this area."

As might be expected, there was considerable divergence of opinion over the philosophy expressed by Dr. Pressman.

Another feature of the 1953 meeting was a look back at the activities and interests of the "first edition" of the Teachers' Section in its period of existence from 1931 to 1942. Dr. Harry Schenck had examined the records of those activities and informed us that an actual movement for an organization to revise and improve graduate teaching began in 1927 when "Dr. Duncan MacPherson of New York City, becoming alarmed at the discrepancies and inadequacies of graduate medical education in otolaryngology and aware of the weakness of a long agitator, arranged by telephone to meet with Dr. George M. Coates in Philadelphia. Dr. Clarence Smith of New York accompanied Dr. McPherson to Philadelphia, and this meeting resulted in the formation of the "Teachers Club for Post-graduate Education in Otorhinolaryngology."

Some others soon became affiliated with this pioneer group and in 1931 Dr. William Wherry proposed that the ideals and objectives of the "Club" deserved Academy support. This resulted in the formation, within the Academy, of a Teachers' Section. This was conceived with enthusiasm and flourished for several years. It served as a forum for discussion on how to improve both undergraduate and graduate teaching, with equal emphasis on the former. Apparently there was no concern at that time about the scope of otolaryngology, and audiology had not had its birth.

In a few years this Teachers' Section apparently "ran out of gas," and it has required the changes in our specialty wrought by the antibiotic era to create a new edition of it.

Today, there would seem to be little doubt but that otolaryngology will effectively solve its problems of adjustment to a changing medical world, and emerge as a specialty stronger than in any pre-

ceding stage in its long history. Some insurance for this can be found in it many provisions for continuation study. No specialty is better fortified with opportunity for graduate clinical advancement with the numerous refresher courses offered by universities from Oregon to Florida or by organizations such as the Research Study Club of Los Angeles.

The basic courses in which surgical anatomy predominates may not find the students these once did before the modern three-year residencies came into being with basic instruction as an integral part. Also, there will probably be less interest in total specialty training by the short course method.

The several societies in otolaryngology equip the specialty superbly with opportunity for clinical progress. Where is there a specialty organization that can duplicate the opportunities of membership in the American Academy of Ophthalmology and Otolaryngology? Then there are the "Triological," the American Otological Society, the American Laryngological Association, and the American Broncho-Esophagological Society, whose annual scientific programs are available to the profession. Added to these are the special groups for advancement in allergy, plastic procedures, otosclerosis, etc., and the many specialty groups on the state and local level, with numerous meetings each year on a monthly basis.

Truly, the opportunities are here and the horizon is almost limitless. What we need are more teachers with vision and the devotion of the quality of a Mosher to the purposes of our specialty.

UNIVERSITY OF MINNESOTA HOSPITALS.

REFERENCES

1. Richards, L. G.: Otolaryngology in Transition; Presidential Address, Laryngoscope 58:455 (June) 1948.

2. McCart, H.: The Future of Our Specialty, Laryngoscope 59:417 (Apr.) 1949.

3. Lederer, F. L.: The Broadened Scope of Otolaryngology, Jour. Internat. Coll. Surg. 13:597 (May) 1950.

4. Lierle, D. M.: The Present Status and Future of Otolaryngology, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 59:796 (June) 1950.

5. Johnston, W. H.: Modern Trends of Surgery and Treatment in Otolaryngology J. A. M. A. 144:977 (Nov.) 1950.

6. The Future of Otolaryngology, Editorial, J. A. M. A. 148:1508 (Apr. 26) 1952.

7. Boies, L. R.: The Future of Otolaryngology, Laryngoscope 52:709 (July) 1952.

8. Roberts, Sam: The Future of Otolaryngology, Letter to the Editor, J. A. M. A. 150:1510 (Dec. 13) 1952.

9. Woodward, F. D.: The Widening Aspects of Otolaryngology, Trans. Amer. Acad. of O. and O. 56:717 (Sept.-Oct.) 1952.

10. Hoople, G. D.: Otolaryngology Looks to the Future, Jour. Med. Educ. 28:29 (May) 1953.

11. Boies, L. R.: Modern Otolaryngology as a Specialty, Minn. Med. 36:368 (Apr.) 1953.

12. Supplement to the Trans. Amer. Acad. of O. and O.: Undergraduate Instruction in Otolaryngology, 1950.

13. Trans. Amer. Acad. of O. and O. 56:493-515 (May-June) 1952.

14. Trans. Amer. Acad. of O. and O. 57:796-822, (Sept.-Oct.) 1953.

15. Trans. Amer. Acad. of O. and O. 58:779-802 (Sept.-Oct.) 1954.

With Wiliam P. Wherry, 1944.   PHOTO WERNER MUELLER

XVI

CARCINOMA OF THE LARYNX AND ITS PRESENT-DAY MANAGEMENT

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Carcinoma of the larynx can be cured in an appreciably higher percentage of cases than at present if these lesions are diagnosed in an earlier stage. Although patients with this condition are now consulting laryngologists more promptly than a decade or two ago, in a number of them the process is already well advanced at the time of the initial visit. Many of them require radical surgical treatment which might well be avoided, if the physician consulted primarily suspects the serious nature of the laryngeal lesion. Most of these neoplasms develop on the vocal cords, and a change in the quality of the voice, or persistent hoarseness, generally occurs early. Accordingly, keener appreciation of the possible significance of chronic hoarseness by the physician and the laity will improve the present situation. Continued education of the public in this connection, too, will help but it is essential also that the physician maintain a high degree of suspicion.

CHARACTERISTIC FEATURES AND DIAGNOSIS

Cancer of the larynx is rather common. An average of 135 patients with this condition are seen annually at the Mayo Clinic. This is approximately 120 patients per 100,000 clinic registrants. Like malignant disease of the mouth and pharynx, it is encountered much more frequently in males than in females, the proportion being more than ten to one. Most of the patients with malignant lesions of the larynx are between 40 and 70 years of age and largely between 50 and 60 years. However, carcinoma of the larynx not infrequently occurs among persons who are less than 40 or more than 70 years of age. In a number of instances, I have seen it during the second and third decades of life and have found it necessary to perform total laryngectomy for its removal in two boys, 16 and 19 years of age, respectively.

As mentioned previously, the earliest and most common symptom of carcinoma of the larynx is hoarseness. This may range from slight huskiness to complete aphonia. Its onset often is insidious but an acute infection of the respiratory tract may appear to induce it. In this connection it must be borne in mind that such lesions at times occur in patients in their teens or twenties. Overlooking this fact may result in loss of valuable time in establishing a diagnosis. Symptoms of advanced laryngeal carcinoma, dyspnea, dysphagia, pain and hemorrhage vary in time of onset, depending on the situation and activity of the growth.

The most important diagnostic measures are a detailed clinical history, painstaking and thorough indirect or mirror examination of the larynx after cocainization if needed and biopsy. Adjuncts are routine laboratory studies including urinalysis, blood count, roentgenographic examination of the thorax, serologic test for syphilis and, if indicated, examination of the sputum. A general physical examination should be carried out routinely. Direct examination of the larynx using a laryngoscope or the suspension apparatus often will yield additional information, especially concerning the extent of the malignant process and degree of fixation. If the tumor has not been traumatized recently and is not acutely inflamed, it usually will have a characteristic, readily recognizable appearance. A specimen can be removed from the most suspicious portion of the lesion at the time of the direct inspection. It should be emphasized that a report of inflammatory tissue returned on a specimen from a lesion that looks malignant clinically demands that further histologic study be carried out. At times the presence of inflammatory reaction in the growth will disguise its identity microscopically as well as grossly. In lymphosarcoma of the larynx repeated biopsies are at times required for diagnosis and it may be necessary to delay removal of tissue for several weeks to permit inflammatory reaction to subside. From experience with more than 1,000 laryngeal biopsies my colleagues and I have observed no harmful effects provided that the procedure was properly performed. It is essential that trauma be minimal and it is advantageous to have the surgeon who is to remove the lesion remove the specimen.

SURGICAL CONSIDERATIONS

Once the diagnosis of carcinoma of the larynx has been established, the most important factor in determining the patient's chance of cure and the possibility of preserving laryngeal function is the experience of the surgeon. The situation, extent, nature and activity of the lesion, previous treatment, especially irradiation, and the age and general condition of the patient must be given consideration.
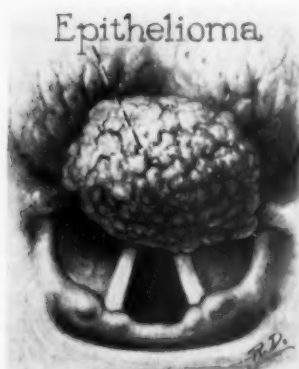
Fig. 1.—Epithelioma of the epiglottis. Lesions in this situation even when more extensive than the one shown here and involving the aryepiglottic folds can readily be removed through the mouth by means of suspension laryngoscopy.

Often a surgeon of limited experience will perform total laryngectomy for a lesion that might have been removed as effectively by a more conservative procedure. While this insures complete eradication of the disease, it does impose a permanent and unnecessary functional handicap.

The therapeutic measures of value in malignant tumors of the larynx include thyrotomy, laryngectomy, hemilaryngectomy or partial laryngectomy, pharyngotomy, endoscopy and irradiation. The first two of these are the procedures generally employed in the removal of such growths. Hemilaryngectomy, pharyngotomy, endoscopic measures and primary irradiation are indicated in only a comparatively small group of selected cases. The proportion of laryngectomies to more conservative methods of removal in any given series of cases of cancer of the larynx varies greatly with the stage of the disease at the time the patients are seen and with the training and experience of the laryngologist. Prior to the past three decades, the great majority of these procedures in most clinics consisted of laryngectomy. In recent years this situation has changed considerably and the converse of this is true in many clinics at present.

Many laryngologists consider that removal of malignant tumors from the larynx by other than an open operation is wholly unjustifiable. In general this is a safe rule to follow. However, many malignant neoplasms situated in the supraglottic portion of the larynx

can be taken care of better through the mouth than by an external operation (Fig. 1). It is possible to see most such lesions satisfactorily with direct, preferably suspension laryngoscopy, and when this is true, preferably transoral removal by means of electrocoagulation is not only feasible but is simpler, safer and just as certain as removal after pharyngotomy.

Most of the supraglottic lesions originate on the epiglottis and secondarily involve the aryepiglottic folds. A few are primary in the latter situation and a few on the posterolateral aspect of the arytenoid cartilage. Those which develop on the epiglottis arise more commonly from the posterior aspect and are usually fungating rather than infiltrating. They tend to grow slowly and metastasize late in contrast to many of the tumors which originate on the base of the tongue and involve the epiglottis secondarily. Because of the slight infiltration, usually present about the base of the former, treatment is effective in eradicating the lesion in a high percentage of cases even when the growth is well advanced. On the other hand, neoplasms which arise in the arytenoid region and pyriform fossae are usually highly malignant and tend to metastasize early. Such lesions should be dealt with through the mouth only if they are seen early and are well localized. Postcricoid lesions usually cannot be seen well enough to permit removal through the mouth.

A small group of patients are not satisfactory subjects for procedures which necessitate any type of direct laryngoscopy for even though complete relaxation is secured by means of a general anesthetic, the anatomic structure of the throat will not permit visualization of the anterior portion of the larynx.

Suspension Laryngoscopy. Suspension laryngoscopy as carried out at present after cocainization of the throat and intravenous anesthesia with pentothal sodium is much easier and safer than it was with the deep ether anesthesia formerly employed. If the lesion under consideration is extensive, preliminary tracheotomy is advisable because of the possibility that sufficient edema may develop postoperatively to produce respiratory obstruction.

The risk associated with removal of laryngeal tumors under suspension laryngoscopy appears to be slight. Generally little immediate postoperative reaction occurs in cases in which this procedure has been used, and convalescence is surprisingly mild and brief. There is little trauma to the tissues aside from that caused directly by the electrocoagulating process. Approximately two and one half to three weeks are required for separation of the slough. When implantation of radium points is used to supplement electrocoagulation,

the local reaction is much more protracted. Bronchopneumonia attributable to aspiration of slough and secondary hemorrhage rarely causes trouble.

Functional results have been satisfactory in these cases. Impairment of the voice or difficulty in swallowing has not occurred after complete removal of the epiglottis together with a portion of the aryepiglottic folds.

Exposure by Laryngofissure. In some instances when malignant tumors in the supraglottic region are infiltrating and extend beyond the epiglottis, removal under laryngeal suspension is not feasible and an open operation, either thyrotomy or pharyngotomy must be carried out. Laryngofissure affords excellent exposure of tumors of the glottis, also of those situated laterally and posteriorly above or below this level but confined within the larynx. Pharyngotomy may be necessary to secure access to infiltrating supraglottic tumors especially those located anteriorly and those involving the hypopharynx and postcricoid region. Many of the latter lesions as well as those of the pyriform fossa require total laryngectomy, and pharyngotomy is performed only very infreqeuntly.

Since it is frequently impossible to determine definitely by clinical examination the limits of the malignant process within the larynx, decision as to the procedure necessary for its removal often must be deferred until direct inspection of the growth is made through a laryngofissure. At the clinic we carry out exploration of this type in all cases in which the growth is not well defined and often it has been possible to remove, through this approach, lesions that on indirect examination appeared to require total laryngectomy. Occasionally an extensive tumor that completely fills the larynx will be found to possess a rather small pedicle with limited attachment so that conservative removal is feasible.

If examination through a laryngofissure discloses that the lesion is too extensive to permit safe removal by this route, laryngeal forceps are applied to the margins of the divided thyroid cartilage and laryngectomy is carried out. As the growth itself is not opened into and traumatized the wound is not contaminated except by laryngeal secretions.

The field of usefulness of laryngofissure has expanded considerably during the past few decades and the attitude of some eminent laryngologists that the operation should be limited to cases of early laryngeal cancer has changed completely. The extent of malignant tissue removable through a laryngofissure varies greatly depending on the situation of the involvement, its nature, activity, extent, and pre-

Fig. 2.—Extensive squamous cell epithelioma, grade 2, that was perforating the larynx and widely infiltrating the surrounding soft tissues in a man 47 years of age. The hyoid bone was removed along with the larynx. Patient is well at present, almost eight years after operation.

vious therapy. It varies also with the training and experience of the physician. Generally, wider experience has demonstrated the feasibility of removing, through a laryngofissure, many malignant tumors of moderate activity or low grade for which laryngectomy was previously performed. On the other hand, increasing experience has in some instances shown the necessity of wider removal of more active growths, particularly when an inflammatory process has been associated or the tumor was recurring.

Sharp excision is rarely depended on alone in removal of cancer of the larynx through laryngofissure even when the growth is small. Electrocoagulation has proved to be an extremely valuable adjunct in this connection and is applied routinely through the laryngeal wound after excision of the lesion. This procedure not only effects

wider removal of the neoplasm but is a guard against possible graft-
ing of malignant cells. In addition, the eschar definitely delays and
often appears to prevent invasion of the freshly opened tissues by the
organisms present in the respiratory tract. Electrocoagulation of
exposed cartilage has not seemed to increase the likelihood of peri-
chondritis. However, most of the coagulated cartilage usually is
removed either by sharp excision or by means of rongeurs in order
to decrease the size of the sequestrum. It is occasionally necessary
to remove sequestrated cartilage later. Aspiration of sequestrated
cartilage, in cases of this type, has not been encountered on our service.
In the hands of experienced laryngologists, this operative procedure
yields five-year cures in more than 80 per cent of cases.

    Laryngectomy. The technique of performing laryngectomy re-
cently has undergone changes in the hands of certain surgeons that
have affected not only the scope of these operations but their effec-
tiveness as well. Some laryngologists have sought to render laryn-
gectomy more conservative by stripping the outer perichondrium
from the surface of the thyroid cartilage thus making the procedure
a subperichondrial resection. The aim of this change has been to
lessen the severity of the postoperative reaction and hasten healing.
Its advocates state that the operation is not suitable when the carti-
lages are invaded or the growth has spread to the muscles and glands
of the neck.

    In my experience, however, involvement and even perforation
of the thyroid cartilage or cricothyroid membrane frequently can-
not be recognized preoperatively by clinical examination, roentgeno-
graphic studies or any other means. In cases in which such involve-
ment is found at operation, opening into the neoplasm is avoided
only by the routine practice of carefully dissecting the muscles from
the anterior and lateral surfaces of the larynx. When the perichon-
drium is stripped from the cartilage in such cases, the neoplasm is
almost certain to be opened. Although this is not disastrous if recog-
nized immediately and the involved region is cauterized thoroughly,
it should be avoided if possible.

    Experience in a number of such cases has convinced me of the
feasibility of effectively removing the lesion even when involvement
of the cartilage and overlying muscles is present (Fig. 2). On num-
erous occasions, I have found limited invasion of the muscles while
these structures were being dissected from the thyroid cartilage. The
involved region was immediately electrocoagulated thoroughly in
order to produce an eschar to protect the wound from the lesion and
then wide electrosurgical excision of the adjacent tissues was carried
out along with removal of the larynx. When such involvement is
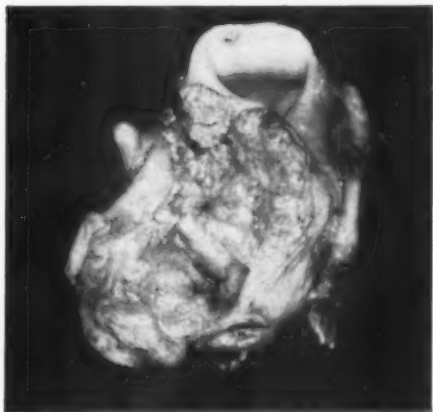
Fig. 3.—Squamous cell epithelioma, grade 2, of left pyriform fossa and cervical portion of esophagus removed from a man 43 years of age. The esophagus was reconstructed with neck flaps three and one-half weeks later. Patient is well at present, approximately two and one-half years after operation.

suspected prior to operation, excision of the overlying soft tissues is done primarily. If necessary all of the muscles, skin and subcutaneous tissues anterior to the larynx are excised. Although the prognosis is less satisfactory than when the larynx is removed prior to perforation, a number of patients have been cured of advanced malignant disease by this means. Carcinoma originating in the postcricoid region, just within the esophageal introitus or in the cervical portion of the esophagus immediately below this may become well advanced and so intimately attached to the exterior of the larynx before it is recognized that total laryngectomy together with removal of the cervical esophagus is required for eradication (Fig. 3). Repair of the esophagus either with a free skin graft or flaps from the neck may be carried out immediately or after a lapse of some weeks or months.

Cases of these types must necessarily be included in any statistical study and they are included in a series of 104 patients undergoing laryngectomy at the Mayo Clinic during the ten years preceding 1944. In spite of this, however, 60.2 per cent of the 88 who were traced lived five years or more.

When palpable enlargement of the nodes of one side of the neck is associated with a malignant process in the larynx, unilateral radical

dissection of the glands and total laryngectomy are performed in a single stage, if feasible. If the condition is not considered surgical because of its extent or the patient's general status, fractional roentgen therapy is advisable. It is still our feeling that in lesions amenable to surgical treatment, operation offers a better outlook than does irradiation.

Hemilaryngectomy. In recent years greater consideration has been given to preservation of function in the surgical treatment of carcinoma of the larynx. Unnecessarily radical operative procedures are now performed less frequently since experience has shown that adequate removal of moderately advanced lesions often can be effected by techniques which do not disturb normal physiologic function. There are definite limits to the extent of tissue that can be removed by thyrotomy or a classic hemilaryngectomy without directly destroying function or causing collapse of the airway. Relining the larynx with a skin graft has been carried out secondarily at times in order to correct the stenosis induced by exceeding these limits. Hemilaryngectomy or partial laryngectomy with immediate skin graft renders it possible to effect more extensive local removal without destroying the voice or disrupting the airway (Figs. 4 and 5). The procedure has now been performed in a sufficient number of cases to permit adequate evaluation and the results warrant its inclusion in the armamentarium of the laryngeal surgeon.

The method was first applied in two carefully selected cases a little more than five years ago and the patients were closely observed for more than a year before further application of the plan was made. It has now been used on the Clinic's service in more than 30 cases. During the same period, thyrotomy has been performed in more than 300 cases and total laryngectomy in a few less than 300 cases for the removal of carcinoma of the larynx. Thus it is obvious that the procedure has only limited application. However, it should be borne in mind that the time under consideration included the period of development of the operation and of observation of results in the initial cases. It seems certain to be carried out in a greater proportion of cases of carcinoma of the larynx in the future.

The operation consists essentially of a radical laryngofissure with removal of as much of the ala of the thyroid cartilage on the side of the principal involvement as is indicated and up to approximately a half of the ala on the opposite side if required. Generally a narrow strip of the superior or inferior border of the thyroid cartilage can safely be left in place to lend support and whenever possible this is done. One half or slightly more of the anterior or anterolateral part of the cricoid cartilage is taken away if necessary. The arytenoid on

Fig. 4.—Squamous cell epithelioma, grade 2, involving the right vocal cord, ventricle of the larynx and ventricular band in a man 40 years of age. The lesion was removed by means of a subtotal laryngectomy and a skin graft was applied immediately to the laryngeal wound. The patient is living and well at present, approximately three and one-half years after operation. He has a good functional voice and no disability.

Fig. 5.—Squamous cell epithelioma, grade 2, involving the entire length of both vocal cords, the anterior commissure and the left arytenoid cartilage in a man 55 years of age. Subtotal laryngectomy removed the lesion and skin graft was done immediately. Patient was alive and well when last heard from two years later.

the involved side has been removed in several of the cases but experience has shown that when this is done, it is advisable to limit removal of the adjacent soft tissues as much as possible; otherwise, difficulty with aspiration during swallowing may occur postoperatively. The involved soft tissues together with the overlying cartilaginous structure are excised en bloc. The trachea is opened prior to hemilaryngectomy or partial laryngectomy so that the respiratory tract below this level can be packed off to prevent gravitation of blood. Fresh frozen microscopic sections of the lesion are checked immediately in order to make certain that adequate clearance of the malignant process has been achieved. Should any of the margins be too narrow, further removal is carried out.

A clean surgical set-up is made. A dermatome graft of moderate thickness is taken from a hair-free region of the chest or abdomen after infiltration with procaine hydrochloride for anesthesia. A stent of sponge rubber is prepared of sufficient size so that it will exert moderate pressure on the entire laryngeal wound when the soft tissues of the neck are closed over it. The skin graft is fastened to the stent with mastisol or rubber cement placed in the laryngeal defect and immediately immobilized by transfixing it with two or three stainless steel wires which pass entirely through the soft tissues of the anterior portion of the neck and through the larynx or upper part of the trachea. The larynx and soft tissues of the neck are closed as in the usual thyrotomy or partial laryngectomy. No drainage is used other than a small iodoform gauze pack below the tracheotomy tube. The ends of the transfixing wires which have been left long are tied firmly over a fluffed gauze pad applied to the wound on the front of the neck.

The postoperative course is mild. After approximately ten days the patient is anesthetized by the intravenous administration of pentothal sodium and the larynx is exposed with a suspension apparatus. The transfixing wires and the ligature attached to the tracheotomy tube are freed and the sponge rubber stent is removed through the mouth. Almost invariably the skin graft will be found to have taken perfectly where it has covered denuded regions. An elastic dilator is made of sponge rubber covered with a condom; this is inserted into the larynx and anchored in position by tying it to the tracheal cannula. It is worn for ten days to two or three weeks, and is then removed through the mouth. A day or two later the tracheotomy tube is lifted out and the tracheal fistula is permitted to close.

The skin graft in the larynx remains thick, dry, and rigid for a variable period following the operation and in some patients ozena results from the accumulation of mucus, debris and crusting. In

Fig. 6.—Appearance of laryngeal skin graft and the adjacent normal mucosa five months following application of the graft. The difference in thickness is striking (hematoxylin and eosin; x30).

the course of time these features tend to undergo spontaneous correction to a great extent, the graft becoming moist, softer and more pliable (Fig. 6).

Patients subjected to this procedure have now been followed for periods ranging from a few months to more than five years. All of the lesions were so extensive that total laryngectomy would have been required if this procedure had not been used. Only one patient had a recurrence of the malignant process. Another also required total laryngectomy because of difficulty with aspiration of fluids after operation. Wide removal of one arytenoid in addition to most of the corresponding side of the larynx had been carried out on this patient. The total laryngectomy was carried out nine months following the partial laryngectomy and at that time no recurrence of the malignant process was demonstrable.

The voice in most of the patients treated in this way has been somewhat hoarser than that after the usual thyrotomy. As following thyrotomy, the voice tends to become clearer and stronger in the course of time but only infrequently is a fair degree of resonance regained after this procedure. In all of the patients the airway has been perfectly adequate.

SUMMARY

More patients with carcinoma of the larynx can be cured, if these lesions are recognized earlier and this is possible if chronic hoarseness is investigated promptly. The most important diagnostic measures are a detailed history, careful mirror examination and biopsy.

The therapeutic measures of value include thyrotomy, total laryngectomy, hemilaryngectomy or partial laryngectomy, pharyngotomy, endoscopy and irradiation. Exploration through thyrotomy often is necessary to determine the extent of the malignant process and accordingly the best means of removal. Many lesions in the supraglottic portion of the larynx can be eradicated with suspension laryngoscopy and electrocoagulation. By means of partial or hemilaryngectomy and immediate skin grafting, some moderately advanced carcinomas with varying degrees of fixation can be cured without disturbing laryngeal function.

Mayo Clinic.

XVII

FENESTRATION – PAST, PRESENT AND FUTURE

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The passing from otolaryngology of one of its great, Dr. Harris P. Mosher, marks also the passing of an era in otologic surgery: with the advent of sulfonamides, and then antibiotics, the bold, life-saving and sometimes destructive postaural mastoid surgery for acute temporal bone suppuration began to be replaced by a new type of endaural surgery under magnification in a clean field to preserve or restore hearing function.

The fenestration operation ushered in and exemplifies the new otologic surgery. This year, the thirtieth anniversary of Sourdille's first successful operation for otosclerosis* is an opportune time to reflect upon the past, to examine the present, and speculate on the future of fenestration.

PAST

With the notable exception of two of Mosher's early colleagues at the Massachusetts Eye and Ear Infirmary, the early history of otosclerosis surgery is European. In 1892 and 1893, Dr. C. J. Blake and Dr. F. L. Jack[1] reported some encouraging hearing improvements on removal of the stapes. The remarks of Jack before the American Otological Society in 1893 are apropos today when stapes mobilization is again in the limelight: "I do not think the results in sclerosis are at all encouraging . . . as far as my reading and experience go, mobilization of the stapes gives results for a few weeks and then the adhesions reform and the trouble returns."[2]

The fenestration operation had its beginning in America with the visit of Maurice Sourdille when he described his work in otosclerosis surgery before the New York Academy of Medicine in 1937.[3] The young, ingenious, and relatively unknown otologist, Julius Lempert, destined to become America's foremost otologic surgeon, performed a month or two later his first fenestration operation.[4] Exactly the

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*In 1947 the author had the privilege of examining and testing the hearing of two patients fenestrated by Sourdille, one in 1932 and the other in 1935. Both patients presented a wide open fenestra clearly visible through the thin skin lining the beautifully healed cavity, with an execellently maintained hearing improvement.
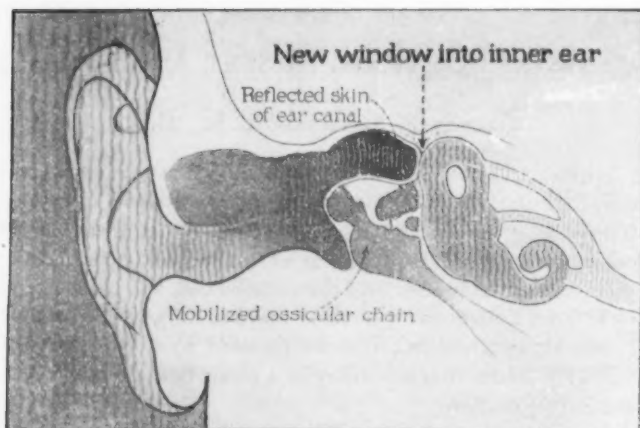
Fig. 1.—Principle of the Sourdille tympanolabrinthopexy utilized in today's fenestration operation.

same procedure was employed as in Soudille's tympanolabyrinthopexy, differing only in the endaural approach, single stage, and use of a rotating burr instead of a scraper to make the fenestra. Today's fenestration operation, except for perfected details in the construction of the fenestra, still employs the basic principles laid down by Sourdille thirty years ago: the passage of airborne sound to the labyrinth is restored by creating a fistula into the horizontal semicircular canal with a plastic skin flap derived from the meatus and attached to the tympanic membrane to cover the fistula and seal the tympanic cavity (Fig. 1).

Like so many important advances in medicine, fenestration at first was greeted with intense skepticism by most, and with open hostility by some. Notable exceptions to the almost universal opposition were two of Philadelphia's most eminent otologists, Dr. George W. Coates and Dr. James A. Babbitt who encouraged Lempert and his handful of early pupils in their pioneer efforts to perfect this new procedure. In 1939 they sponsored the formation of a small study and discussion group that has since grown into the Otosclerosis Study Group of the American Academy of Ophthalmology and Otolaryngology.

From the first, Lempert encouraged young otologists to study the difficult technique of the operation on the cadaver under his personal supervision. From a small beginning Lempert's course grew

into the fountain of inspiration for many hundreds of otologists from America and abroad. As a result of this monumental teaching effort, as well as the increasing numbers of successful operations, the opposition has all but disappeared. Today fenestration is probably the most frequently performed major operation upon the ear, and is well known and accepted for its fine results, in properly selected cases, for otosclerosis.

The development of fenestration surgery after its introduction to America in 1937 has been toward improved technical details especially to prevent osteogenic closure, and toward a clearer appreciation of its limitations and a better definition of its indications.

Like Holmgren and Sourdille, Lempert and his early pupils soon found that the majority of fenestration operations failed to maintain the hearing improvement due to osteogenic closure of the fenestra. Two improvements in technique were soon introduced which appreciably diminished the percentage of closures to about 25 per cent of operations. At one of the early meetings of the Otosclerosis Study Group. Wm. A. Wagner[5] reported several cases with good initial hearing improvements after removal of the incus, and suggested that perhaps the incus did not have to be left in its normal position attached to the stapes as advocated by Sourdille and Lempert. In 1941 Lempert[6] described the "nov-ovalis" technique where the incus was deliberately removed and the fenestra was made farther forward into the wider ampulla of the horizontal semicircular canal.

Meanwhile another member of the Otosclerosis Study Group[7] had found that by continuous irrigation while making the fnestra bone dust could be more effectively washed away, with fewer closures. The operating microscope, first introduced for otosclerosis surgery by Gunnar Holmgren some years before, was revived for the creation of a more perfect fenestra free from bone dust and chips.[7]

The next few years saw a number of ingenius modifications to further prevent osteogenic closure. These included a conjunctival graft laid over the fenestra beneath the tympanomeatal skin flap; cautery or irradiation of the bone around the fenestra; a metal obturator fitted to the margins of the fenestra; lead rubbed onto the bone around the fenestra.[8] Especially promising for a time was the use of a cartilage plug inserted into the fenestra, an idea first conceived and used on the experimental animal by Philip MacDonald[9] and subsequently adopted by Lempert.[10] One by one each of these modifications was abandoned as its disadvantages were found to outweigh its advantages.
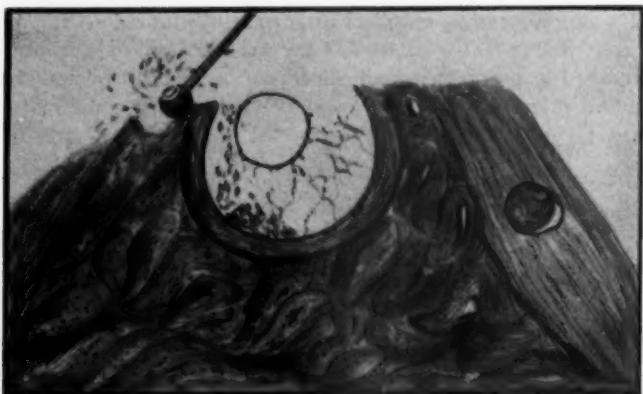
Fig. 2.—Biologic principles governing osteogenesis utilized to prevent osteogenic closure of the fenestra. Bone dust, a major cause for closure in the original operation.
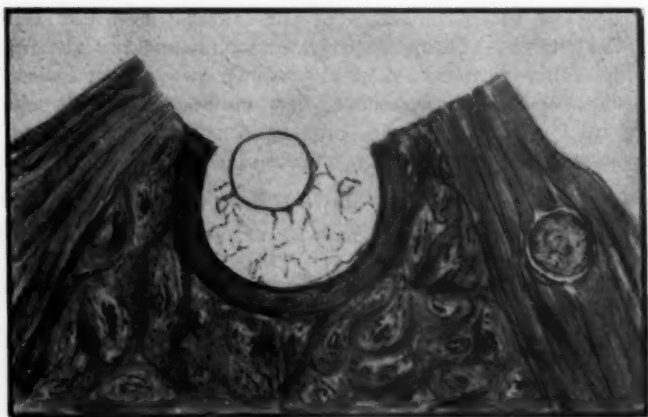
Fig. 3.—Biologic principles governing osteogenesis utilized to prevent osteogenic closure of the fenestra. Type of fenestra formerly constructed without wide exposure of the endochondral layer.
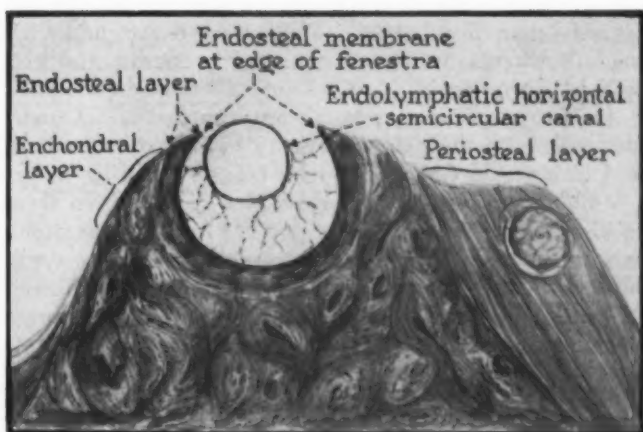
Fig. 4.—Biologic principles governing osteogenesis utilized to prevent osteogenic closure of the fenestra. Fenestra enchondralized. Bone dust removed by irrigation. Endosteal membrane preserved to sharp edge of fenestra.
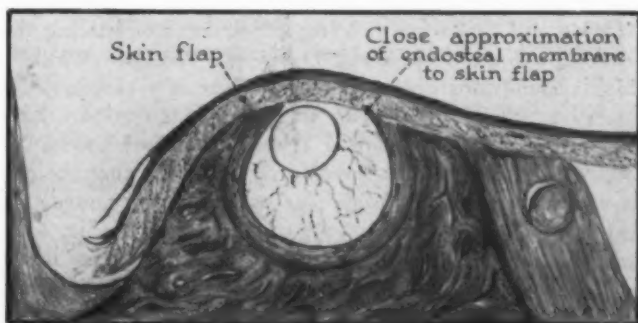
Fig. 5.—Biologic principles governing osteogenesis utilized to prevent osteogenic closure of the fenestra. Periosteum of skin flap in contact with endosteum to produce healing of the bone by first intention.

Meanwhile, experimental research on the ear of the laboratory animal was under way to study the biologic factors that influence osteogenesis after fenestration. These were begun under Gunnar Holmgren's direction by Hans Engstrom[11] in Sweden, and were first undertaken in America by Norton Canfield[12] and Edmund P. Fowler, Jr.[13] In 1940 an extensive research program that is still under way was initiated at Northwestern University[14] followed by similar studies at the University of Chicago by John Lindsay[15] and by Julius Lempert, and his associates[16] in Boston and New York. From these basic research studies the nearest answer thus far found to the problem of osteogenic closure has gradually emerged. The clinical application to the operation of the biologic factors that influence osteogenesis (Figs. 2 to 5) has finally reduced the failures due to closure to less than five per cent and in some studies to around one per cent of operations.[17]

PRESENT

As so often happens to new developments in medical science, the early opposition was followed by a period of over-enthusiasm with many poor results by surgeons inadequately trained in the precise technique of fenestration, or from operations on improperly selected cases. The improved results today are attributable to a clearer appreciation of its limitations, and better selection of cases, as well as to improvements in technique.

The first limitation of fenestration surgery is that for satisfactory results it requires special training beyond the residency, and is not a procedure for the occasional operator.

The second limitation concerns the amount of hearing that can be restored. In 1950 Hallowell Davis and Theodore Walsh[18] made the highly significant observation that there is a certain residue of unrestored conductive loss after successful fenestration. The next year a statistical study[19] showed that this residual conductive loss averages 25 decibels for the speech frequencies, so that the probable result of fenestration in a particular patient may be predicted as a hearing level 25 decibels below the cochlear reserve (Fig. 6). This means that in partial stapes ankylosis with a conductive loss of less than 35 decibels fenestration cannot be expected to give a satisfactory hearing improvement of more than 10 decibels for the speech frequencies. The ideal case for fenestration will have not only normal cochlear function but a maximum conductive loss of 50 to 60 decibels.

A further aid in selecting cases for fenestration was the demonstration by Carhart[19] that there is a slight shift in the bone audiogram in stapes ankylosis which largely disappears after fenestration
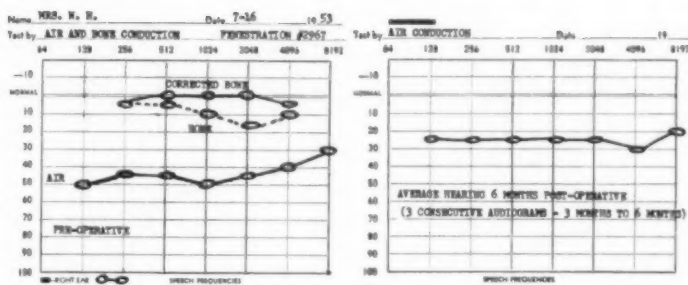
Fig. 6.—Audiograms in a case of otosclerosis without cochlear less with average result from fenestration.

and therefore is probably of mechanical rather than neural origin. This shift takes the form of a shallow notch, deepest at 2000 cycles. The best estimate of the cochlear reserve in a case of stapes ankylosis is the bone conduction audiogram corrected for this "Carhart notch" by 5 decibels at 512, 10 decibels at 1024, 15 decibels at 2048 and 5 decibels at 4096 cycles (Fig. 6).

The question of fenestration of patients who fall short of ideal suitability is still being debated. Comparative measurements of a patient's actual hearing ability for speech, while wearing his hearing aid at its accustomed comfort setting, with his ability to hear and understand speech after fenestration, have helped to solve this question.[19,20] It has been found that on the average patients of ideal suitability hear slightly better without an aid after fenestration than with their aid before operation, with this exception: in quiet surroundings where added acuity may be needed the hearing aid can be turned up in loudness, while the fenestrated ear cannot.

In patients showing early, mild cochlear losses in their bone conduction tests along with complete stapes ankylosis and a large air-bone gap, similar measurements show that in some cases the hearing for speech after fenestration exceeds the hearing with the patient's hearing aid, but that on the average it is a little poorer in volume though a little better in clarity (discrimination) than with the hearing aid. As the degree of cochlear nerve involvement increases, the hearing result by fenestration decreases relative to a good hearing aid.

The final decision for or against fenestration must be individualized taking into account not only the anticipated hearing result predicted from the hearing tests, but the social and economic hearing
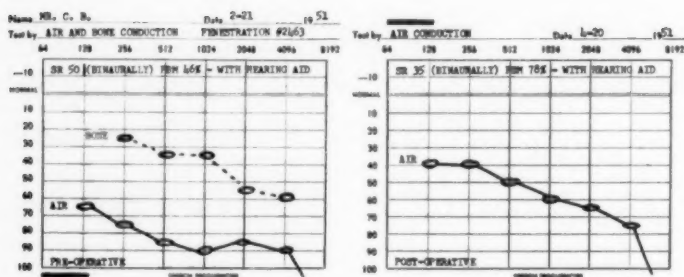
Fig. 7.—Audiograms in advanced otosclerosis with considerable cochlear nerve deterioration fenestrated to improve the use of a hearing aid. Left: air and bone audiogram with speech reception (SR) threshold and discrimination score PBM) with hearing aid. Right: Audiogram two months after fenestration with SR and discrimination score with hearing aid.

needs of the particular patient, including his ability to accept and adjust to a hearing aid.

In cases of otosclerosis of ideal suitability with complete stapes fixation and normal cochlear function for the speech frequencies, fenestration is the rehabilitative procedure of choice, provided the patient is told and accepts the slight but definite risk of failure.

In less ideal cases fenestration may be advisable for patients who are unable to adjust to a hearing aid, provided they are told and accept the definite limitation of improvement imposed by their impaired cochlear function.

In rare cases of profound otosclerotic deafness, fenestration may be justified to improve the use of a hearing aid, with no thought or hope of restoring practical unaided hearing (Fig. 7).

FUTURE

Probable future developments in fenestration surgery will include improved methods for preventing postoperative serous labyrinthitis, techniques for overcoming the residual conductive loss remaining after successful fenestration, and more definitive information regarding the permanence of the restored hearing.

Today's most frequent cause of failure appears to be postoperative serous labyrinthitis. Experimental animal research demonstrates the role of hemorrhage into the labyrinth and infection of the cavity as contributing factors to this inflammation of the labyrinthine con-

tents. With the utmost care to maintain perfect aseptic technique at the operation and during the first month of postoperative treatment, and routine antibiotic therapy, infection of the wound is virtually never seen, and healing by epidermization occurs without granulations or suppuration. Nevertheless serous labyrinthitis still causes about five per cent of operations to fail, perhaps due to factors not yet known.

In 1948 Arthur Juers demonstrated a slight further gain in hearing by blocking the tympanic membrane in the fenestrated ear.[21] This and other methods for enhancing the sound pressure differential between the fenestra and the round window[22] hold the promise of future reductions in the residual conductive loss after fenestration.

Studies of the permanence of the hearing improvements after fenestration are facilitated by a "fenestration score board" which keeps a current record of the hearing results of large numbers of operated cases with periodic audiograms year after year. Inasmuch as fenestration merely by-passes the otosclerotic lesion at the oval window and does nothing to alter the underlying bone disease, it has been assumed that even if the conductive loss of stapes ankylosis is permanently reduced by the new labyrinthine window, the progressive cochlear deterioration so common in otosclerosis will eventually rob the patient of his restored hearing.

A few studies of long-term results are now available. One of these[17] includes 390 cases with audiograms five to ten years after operation. In 273 or 70 per cent the hearing improvement had been maintained at a stable level so that the average loss for the speech frequencies for these cases (including ideal cases and cases with preoperative cochlear deterioration), was 34.0 decibels one year after operation and 34.5 decibels in the most recent test five to ten years after operation (Fig. 8). This suggests that in many cases the underlying otosclerotic bone disease becomes inactive and the hearing becomes stabilized.

It is illogical to expect fenestration to retard cochlear nerve deterioration in cases of otosclerosis, yet there is some preliminary evidence[23] suggesting that in some cases better cochlear nerve function is maintained in the fenestrated ear than in the opposite unoperated ear. This unexpected (and still questionable) benefit of the operation needs to be confirmed or disproved by future long term studies.

Speculation regarding the future of fenestration versus stapes mobilization must remain as pure speculation until more is known

Fenestration, Past, Present & Future
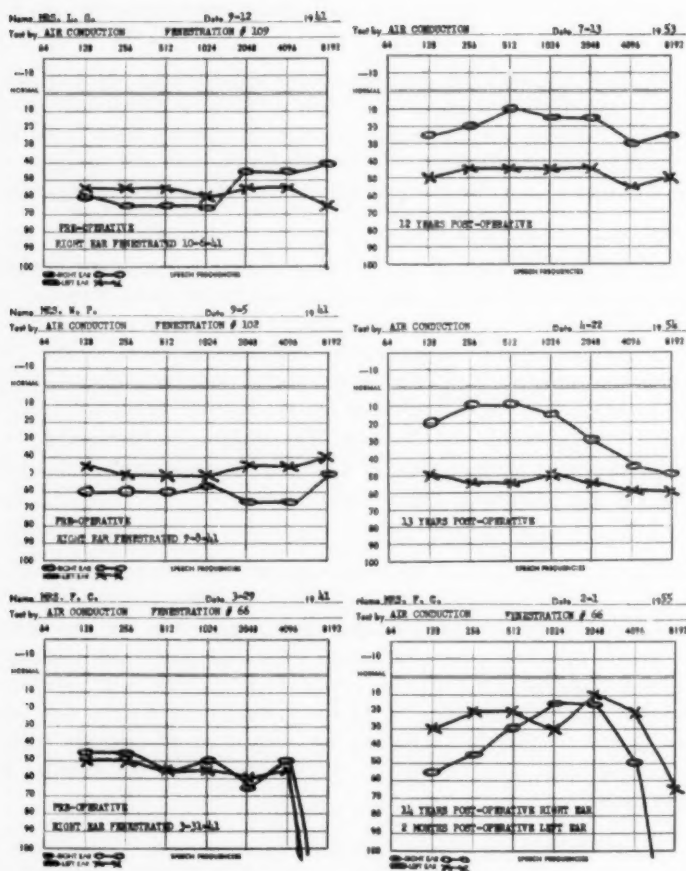
Fig. 8.—Audiograms in three cases with improvement maintained 12, 13 and 14 years after fenestration.

of the latter procedure. It is conceivable that the simpler mobilization may prove to be an optional preliminary procedure for certain cases of incomplete stapes ankylosis. Considering the solid mass of otosclerotic bone that frequently replaces the stapes footplate and oval window, stapes mobilization cannot conceivably replace fenestration in cases of advanced stapes ankylosis. Meanwhile, the otologist should maintain an open mind tempered by a healthy scientific skepticism, toward this and other new procedures for otosclerosis.

55 EAST WASHINGTON ST.

REFERENCES

1. Blake, C. J.: Middle Ear Operations, Trans. Amer. Otol. Soc. 5:2:306, 1892.

2. Jack, F. L.: Further Observations on Removal of the Stapes, Trans. Amer. Otol. Soc. 5:3:474, 1893.

3. Sourdille, M.: New Technique in the Surgical Treatment of Severe and Progressive Deafness from Otosclerosis, Bull. N. Y. Acad. of Med. 13:673, 1937.

4. Lempert, J.: Improvement of Hearing in Otosclerosis—A New One-Stage Surgical Technique, Arch. Otolaryng. 28:42, 1938.

5. Wagner, Wm. A.: Unpublished Communication to Otosclerosis Study Group.

6. Lempert, J.: Fenestra Nov-Ovalis: A New Oval Window for the Improvement of Hearing in Cases of Otosclerosis, Arch. Otolaryng. 34:880, 1941.

7. Shambaugh, G. E., Jr.: A Modified Fenestration Technic—Analysis of Results in One Hundred and Forty-one Consecutive Operations, Arch. Otolaryng. 36:23, 1942.

8. Lempert, J.: The Permanently Patent Fenestra Nov-Ovalis, Laryngoscope 61:215, 1951.

9. MacDonald, Philip G.: First Experimental Use of Cartilage to Maintain Patent Fenestra in Bone, Arch. Otolaryng. 41:241, 1945.

10. Lempert, J.: Lempert Fenestra Nov-Ovalis with Mobile Stopple, Arch. Otolaryng. 41:1, 1945.

11. Engstrom, Hans: Über das Vorkommen der Otosklerose, nebst Experimentellen Studien über chirurgische Behandlung der Krankheit, Acta Otol. Suppl. 43:153, 1940.

12. Canfield, N.: Labyrinthine Fistulas; Report of Experiments in Vital Responses to Various Methods of Producing Defects in Bone, Arch Otolaryng. 30:50, 1939.

13. Fowler, E. P., Jr.: Closure of Operative Fenestra in Labyrinth, Arch. Otolaryng. 34:209, 1941.

14. Shambaugh, G. E., Jr.: Fenestration Operation for Otosclerosis—Experimental Investigations and Clinical Observations, Acta Otolaryng. Suppl. 79, 1949.

15. Lindsay, J.: Histologic Observations on Healing of Labyrinthine Fistulas in Monkeys, Arch. Otolaryng. 43:37, 1946.

16. Lempert, J., Meltzer, P. E., Schall, L. A. and Wolff, D.: Osteogenesis Following Fenestration of Vestibular Labyrinth of Rhesus Monkey: Controlled and Experimental Study, Arch. Otolaryng. 46:512, 1947.

17. Adin, Louis E., Jr. and Shambaugh, G. E., Jr.: A Study of Long Term Hearing Results in Fenestration Surgery, Arch. Otolaryng. 53:243, 1951.

18. Davis, H. and Walsh, T. E.: The Limits of Improvement of Hearing Following the Fenestration Operation, Laryngoscope 60:273, 1950.

19. Shambaugh, G. E., Jr. and Carhart, R.: Contributions of Audiology to Fenestration Surgery, Arch. Otolaryng. 54:699, 1951.

20. Shambaugh, G. E., Jr.: Comparison of Improvement in Hearing Following the Fenestration Operation With That Obtained by Wearing a Hearing Aid, Arch. Otolaryng. 41:189, 1945.

21. Juers, A. L.: Observations on Bone Conduction in Fenestration Cases: Physiological Considerations, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 57:28, 1948.

22. Shambaugh, G. E., Jr.: Technic for Increasing Sound Pressure Differential Between Fenestra and Round Window to Enhance Hearing Improvement Following Successful Fenestration, Trans. Am. Acad. of Ophth. and Otolaryng., 1949.

23. Shambaugh, G. E., Jr.: Effect of Fenestration Operation on Nerve Degeneration of Otosclerosis, Trans. Am. Academy of Ophth. and Otol., 1949.

XVIII

A PRIMARY CHOLESTEATOMA OF THE MIDDLE EAR

CASE REPORT

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Cholesteatoma verum or a true congenital cholesteatoma as a tumor of the temporal bone is quite rare. Until recently I never had observed such a case though I had seen many so-called silent cholesteatomas which had developed through a defect in the ear drum or Shrapnell's membrane. The present case is being reported as probably representing a true congenital type of tumor. It is of special interest because it presented several problems as to proper management.

A 45 year old woman consulted me because of stuffiness in her right ear of about two months' duration. For the past month she noticed that the hearing in that ear was not as acute as that of her left ear. Past history was completely negative except that her ears sometimes felt full or stuffy after flying. On examination the left ear was negative. The posterior half of the right drum was somewhat distended and had a yellowish appearance. The drum was fixed on testing with a pneumatic otoscope. The anterior part of Shrapnell's membrane was not depressed but there was a questionable pin-hole perforation present. Nose, throat and nasopharynx were negative.

Impression. Non-infected cholesteatoma of right attic and middle ear probably arising from a defect of Shrapnell's membrane.

X-rays of her mastoids were ordered and she was scheduled for operation a week later. To my surprise the x-rays revealed normally developed mastoids which were well pneumatized. There was no evidence of cellular necrosis or of osteosclerotic change. X-ray report: Normal mastoids.

The patient was examined again the day before she was scheduled for operation. During the intervening week the right drum had become distended further and the posterosuperior quadrant showed a beginning necrosis though there was no perforation. The patient stated that she had felt a dull aching sensation in the ear for the past few days. She was admitted to the hospital for operation the following day.
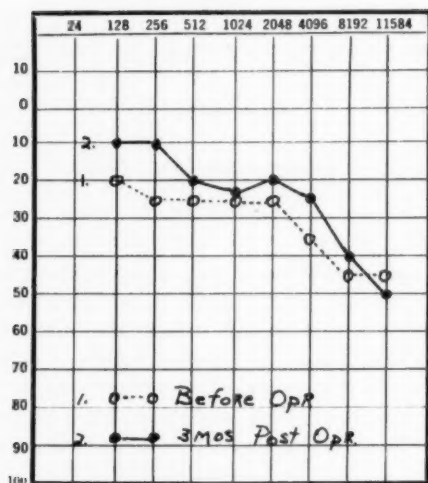
Fig. 1.

Question. 1. Supposing this to be a cholesteatoma, is a radical mastoidectomy indicated? Here is a large pneumatic mastoid with no radiographic indication of pathology. It is well known that a radical cavity in a pneumatic mastoid heals slowly and poorly. 2. Can this patient's hearing be preserved at a practical level? Her hearing loss in the affected ear is only 25 decibels throughout the speech range.

It was decided to perform an atticotympanotomy and, if necessary, to extend the operation as indicated by the operative findings. By way of the endaural approach the membranous lining of the external meatus was elevated as far as the annulus and Ravinian notch. The outer attic wall was removed with curettes and drills. Cholesteatoma could be seen mounding up from the middle ear external and medial to the incus. The posterior half of the drum was then freed from the annulus and turned forwards revealing the middle ear to be filled with cholesteatoma. This was removed mostly with suction. The long process of the incus was found to have been partly absorbed and disconnected from the stapes, so the incus was removed and also the head of the malleus. The attic appeared to be lined with healthy mucosa. Shrapnell's membrane was found to be intact. The stapes was surrounded by cholesteatoma, and the footplate tilted downwards by a mass of cholesteatoma between the oval window and

the Fallopian canal. With magnified vision it was possible to remove the lining of the cholesteatoma sac from the middle ear and internal surface of the ear drum revealing healthy looking mucosa beneath it. The only area where the mucosa was absent was on the posterior surface of the middle ear in the region of the pyramidal eminence. The bone in this area was superficially curetted as being the only area where a cholesteatoma matrix might remain. A tympanomeatal flap was then fashioned and turned upwards and inwards to line the inner attic wall and seal off the aditus. This was held in place with paraffin mesh gauze. The posterior half of the ear drum was packed against the inner wall of the middle ear with two purposes in view, first, to place the drum in contact with the head of the stapes in the hope of a better hearing level and, secondly, to exteriorize the posterior wall of the middle ear from which the cholesteatoma seemed to arise.

The postoperative convalescence was rapid and uneventful. The wound healed by primary intention. Upon removing the packing on the fifth day, a small pin-hole perforation was noted in the drum in the posterosuperior quadrant in the area which had appeared to be necrotic prior to surgery. This perforation has persisted and appears to serve as a vent for the aditus and mastoid. After the packing was removed at no time was there any discharge or secretion from the middle ear. The posterior segment of the drum appears atrophic and is adherent to the head of the stapes. The patient has remained symptom free and states that her hearing is better than before operation (Fig. 1). Four months have elapsed since surgery.

Criticism. Was it not a mistake to attempt to seal off the attic and aditus with a meatal flap? If this had not been done would the perforation of the drum have occurred?

<div align="center">SUMMARY</div>

1. A case of probable cholesteatoma verum of the middle ear is reported arising from the area of the pyramidal eminence.

2. The operative procedure for handling this case has been discussed and criticized. The mastoid process was not opened. A perforation of the drum resulted which might have been prevented.

3. Hearing has improved over the preoperative level.

4. An attempt to preserve the ossicular chain in this case would have resulted in a failure to eradicate all the pathology.

121 UNIVERSITY PLACE.

XIX

THE MANAGEMENT OF THE CHRONICALLY
DISCHARGING EAR REQUIRING RADICAL
MASTOIDECTOMY

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Little that is new can be said about the radical mastoid operation in its fundamental concept. In the early volumes of the Transaction of the American Otological Society and in later accounts of the Proceedings of the New York Academy of Medicine one finds ample evidence that the basic principles of radical mastoid surgery were recognized by such American pioneers as Dench and Whiting of New York, Randall of Philadelphia, Orne Green of Boston. Basic rules of procedure were later set down for us by men like Richards and Morisette Smith, to name only two.

Reading what Dench and Randall, for example, had to say about the radical operation back in the nineties, one feels a tremendous admiration for the keen perception, the vision, the sound judgment that illuminated their thinking. And they had courage, too. We must remember that even the simple mastoidectomy introduced by Schwartze in 1873 had been a burning issue, the conservative element in American medicine becoming reconciled to it only after more than a decade of hostile opposition and acrimonious debate. Some of these die-hards, clinging to their leeching and blue ointment, were still fighting a last-ditch battle against the Schwartze procedure when European surgeons, recognizing the need for a more radical operation in cases of chronic suppuration, got to work on a technique for more extensive removal of diseased tissue. Following in the footsteps of Kuster, who was the first to remove the posterior wall of the meatus, and von Bergman, the first to remove the outer attic wall, Zaufal and Stacke—working independently, finally evolded the procedure we now call the radical mastoidectomy, or tympanomastoidectomy. The 'nineties had just begun.

What conservative reaction to this new procedure was in America one can easily imagine. Men like Dench and Randall, on the other hand, at once realized what the radical operation could accomplish, seized upon it with alacrity, used it, spread its virtues with missionary

zeal among their colleagues, championing its cause everywhere against all opposition. They were quick to formulate some basic principles.

"A man must know healthy bone from diseased bone," observed Dench, urging really thorough exenteration, "and he must be prepared to remove all diseased tissue. Therein lies the secret of all successful otology."

His contemporary, Randall, whose zeal for thoroughness had already earned him the reputation in his native Philadelphia of being a "bloodthirsty" surgeon, early recognized the necessity for exercising meticulous care.

"Success," he said, "depend upon attention to the minutest detail, not only throughout the operation itself but in the entire after-treatment."

Here we have some basic truths about the radical procedure that hold as surely for us today as they did when first stated more than a half century ago.

DIAGNOSIS AND CHOICE OF PROCEDURE

I cannot refrain from quoting, again, and for the same reason, namely, because it has never been better said: Lillie's words are, "The information obtained from a detailed history is certainly as important as the mastoid surgery."

One cannot too often or too emphatically stress the importance of eliciting every possible detail concerning the patient's symptoms. Such details as—the character of the discharge, its duration, whether it is continuous or intermittent, whether there is vertigo, headache, chills, hearing loss. Important as these matters are, a discussion of them here, in this context, appears to me to be too elementary. Nor should it be necessary to discuss in detail the many and varied physical findings associated with the multitudinous pathologic processes one meets with, in chronic suppuration. Any number of texts provide excellent plates one can study for this purpose.

My main object will be to discuss on what basis the surgeon decides to carry out a radical procedure, and to what extent it should be a complete radical procedure in the classic sense.

Surgery for chronic suppuration has gone beyond the point where the only considerations are to eradicate the disease, prevent complications and stop the discharge. Today the competent surgeon goes one step farther and tries to conserve or improve the hearing. It can and should be done, wherever possible. It is with the con-

servation of hearing that I have particularly concerned myself, and it is about this aspect of radical mastoid surgery that I wish to deal.

Part of our problem would be greatly simplified if the diseased process would show greater respect for anatomic boundaries. If it would only confine itself to the middle ear proper or keep itself neatly limited to the epitympanum, antrum and mastoid, the surgeon's life would be a happier one. But it does no such thing.

Morrisette Smith very simply yet adequately divides cases of chronic otitis into three classes: 1) intratympanic infection, with little or no involvement of the attic; 2) disease involving not only the tympanum but the attic, antrum and, to a limited extent, the mastoid; 3) disease involving the intratympanic structures and structures of the attic, antrum and mastoid as well.

Let us consider specific instances of these classifications, as they bear on the choice of operative procedure: where a complete radical is called for, and where, to conserve hearing, it is just as clearly indicated that the mucosa over the promontory and regions of the windows be left intact.

SPECIFIC CASES

First, there is the ear in which the disease appears to be confined to the tympanum, with either an anterior or central perforation. We are all familiar with the widely held view that this situation rarely calls for surgical interference, a view that definitely belongs in the "department of dangerous generalization." Disease causing such perforations often does extend into the uppermost regions of the middle ear, antrum and mastoid, giving rise to a progressive osteitis that will eventually require an extensive surgical procedure. When that time comes, I carry out a radical procedure as regards the epitympanum, antrum and mastoid but I conserve the middle ear mucosa. If it appears from the history that respiratory infections do not exacerbate the discharge, I leave the eustachian tube alone. The hearing in these patients is usually good and should be conserved.

Another case in point is that of the chronic ear with considerable destruction of the drum, just a remnant of malleus remaining; the mucosa of the middle ear is thickened. The hearing may be very poor, or reasonably good. The disease would appear to have destroyed the middle ear in the initial stage of active infection, persisting in the epitympanum, antrum and, possibly, the mastoid. If the hearing is poor, I make no effort to conserve any of the mucosa in the middle ear but carry out a complete radical. If the hearing is reasonably good, I make every effort to handle the middle ear mucosa as deli-

cately as possible so as not to injure it, particularly if there is hearing loss in the opposite ear.

Still another type of chronic ear calling for specific action is the one in which the suppuration has failed to respond to adequate middle ear medical or minor surgical management. The middle ear will be found to show marked changes in the mucous membrane. The tissue is highly vascularized. Granulations or polyps may be present. The hearing is usually poor. No evidence of cholesteatoma is apparent, but one does find signs of osteitis in the epitympanum, antrum and mastoid. I doubt if in this type of case anyone would quarrel with the decision to carry out a complete radical operation from tube to mastoid tip.

Now, what to do with the chronic ear that shows a well-epithelialized medial wall, but definite activity in the regions above? Often one finds remnants of drum firmly bound to the medial wall. It is evident that the eustachian tube and middle ear space are not the source of the discharge. Should one remove this evidence of a healed process? Certainly not, if serviceable hearing is present; but definitely so if the hearing is not enough to be worth bothering about.

Next are the cases with specific cholesteatoma problems, where the disease is predominantly in the epitympanum, antrum and mastoid. The middle ear is not seriously involved, yet the drum and ossicles are. There is but one solution; complete eradication of all diseased tissue. If hearing is at all serviceable, however, I leave the tympanic mucous membrane, for the reason that the disease process is above the middle ear proper.

Cholesteatoma may be confined to a limited area or extend over a larger one. In most instances, it is circumscribed. The cavities are smooth-walled or only slightly irregular, with very little cholesteatomatous prolongation into channels remote from the main mass. This type of cavitation permits the surgeon to carry out a classic radical procedure; the cavity of the middle ear, epitympanum and mastoid is ideally shaped and usually not too large. Cholesteatoma may, on the other hand, be invasive, usually in mastoids with pre-existing pneumatic spaces, reaching into recesses beyond the confines of the antrum and perilabyrinthine region, and extending even into the tip and marginal areas. A huge cavity is created, posing an aftertreatment problem. These are the cases for which cavity-obliterating procedures have been designed.

I have been able to cover only some of the various types of cases calling specifically for either a complete or a conservative radical operation, depending on whether it is possible or not to conserve hear-

ing. I have tried to make clear that the case in which I depart from
the classic concept of the complete radical (exenteration of all dis-
eased tissues from stem to stern, so to speak) and exercise the utmost
care to preserve the mucosa on the medial wall of the tympanum, is
the one in which hearing, in spite of the chronicity of symptoms, is
reasonably good. It is good because there is good mobility of the
stapes and round-window membrane, and this function must be pre-
served if hearing is to be preserved. What makes me firm on this
point of leaving the mucosa alone is repeated observation that where
hearing is good to begin with, complete stripping of mucosa from
these areas markedly reduces it. The conservation of mucosa is par-
ticularly advisable in bilateral deafness.

It has been said that if one removes all mucous membrane from
the medial wall and skin-grafts the surface, the hearing can be re-
tained and often even improved. This has definitely not been my
experience. The only time I have observed good hearing in a patient
with a skin graft over the medial wall was when, for some unexplained
reason, an air space developed under the graft in the region of the
windows.

Now, some of you will say that this kind of fence-straddling
(leaving mucosa or scarred, adhesive membrane over the promontory)
constitutes, in effect, a modified radical. I do not see how one can call
a procedure a modified radical in which ossicles are not retained. I
prefer to call it a conservative radical operation. It is nonetheless
thorough, for all that.

It might be well, at this juncture, to particularize my concept
of what constitutes good hearing. Occasionally patients with chronic
suppuration requiring radical mastoidectomy have hearing for pure
tones at 30 db in the speech frequencies. Every effort should be made
to conserve hearing in these instances. These individuals hear nor-
mal conversation at distances of 10 to 12 feet. Others, with a loss
of from 35 to 40 db, hear conversation at 3 to 6 feet. Patients with
a loss of 40 to 50 db, can hear normal conversation at distances of
2 to 4 feet. Those with a loss greater than 50 db, the chances are,
will have little or no hearing for normal speech. These are approxi-
mate figures. If the higher frequencies are poorer than the speech
frequencies the hearing is usually poorer than I have estimated.

Reports have been published claiming hearing of normal con-
versation at distances of 20 or more feet in patients who have had a
complete radical mastoidectomy. Allowing for a rare exception, I
doubt that, had modern audiometric methods for pure tones and
speech been used in the testing, such spectacular results would have
been achieved.

OPERATIVE PROCEDURE

Instruments. It remains now to discuss actual operative procedure. Modern endaural surgery calls for modern instruments. Some 70 years ago, Dr. Gruening declared, "What I can do with the drill, I can do with the chisel, and what I can do with the chisel, I cannot do with the drill."

Were he alive today to see what can be accomplished with the modern electric drill and burrs, he would probably be heard to say something much more like: "What I can do with the chisel I can do better with the drill; and what I can do with the drill, I cannot do with the chisel."

The curette, on the other hand, has lost none of its usefulness over the years. Those of us who have been initiated into the use of the long-shanked, long-handled curette favored by Lempert find it particularly well suited to endaural radical surgery. The various steps of the procedure, particularly the widening of the osseus canal, have been immensely facilitated and the surgery made safer by the use of the electrically driven dental motor with its burrs of various cutting surfaces.

As for accessories like the electric headlight and loupe, those of us who have known what it was to work without them can appreciate to the full the advantages they afford the surgeon in carrying out the more delicate steps of the radical operation.

Incisions: Since every operation must begin with the incision, let us at this point consider the virtues of those most widely in use. In the early years only one incision, the postauricular, was employed, and I am sure that all but the youngest surgeons practicing today have used it. We knew of no other. The men who originated the radical procedure realized that, having made a large cavity, they would have to have access to it. So there soon broke out a rash of cleverly designed incisions of the canal wall membrane and external meatus. You all know, I am sure, the Korner, Siebenmann, Panse, Passow, Bruhl and Stacke flaps—to name but a few. Most of them accomplish their purpose.

In recent years the endaural incisions of Lempert have found increasing favor. I do not argue the virtues of the postauricular versus the endaural incisions. I think that I need only say after more than 20 years of using the postauricular incision, I have gone over completely to the endaural, which I have used exclusively for the past 15 years. I have done so on the basis that the incision of choice is the one that ensures 1) minimal trauma to surrounding soft

tissues; 2) maximum exposure of the canal walls, middle ear and mastoid; 3) ease of instrumentation, whether one uses curette, gouges or motor-driven burr; 4) an adequate external orifice to simplify after-care; and 5) the best cosmetic result.

My experience has proved the endaural approach to accomplish these things. This is where I stand on the question of incisions.

Rules of Procedure. Before the surgeon busies himself with the incisions, he must already have firmly fixed in his mind a concept, and a clear one, of what constitutes a good radical cavity. He must not be satisfied with merely breaking through the bridge, clearing out what he can without danger, and then leaving the operating room satisfied that he has done his best according to his lights. This man, if he does end up with a good result, has achieved it more by luck than by intent.

At the very beginning of this discussion, I mentioned that the rules of good procedure in the radical mastoidectomy were laid down for us long ago by Richards. Paraphrasing his statement, which was in the form of a list of the specific errors that lead to a bad result, one arrives at the following:

The approach to the tympanum through the external bony canal must be adequate.

That portion of the tympanic bone that forms the anterior, inferior and posterior portions of the canal must be reamed so that it has a cylindrical form, without angulations.

The canal and tympanum should be almost one diameter in all its axes.

If this procedure is followed, there is no chance for infection to develop in the hypotympanum and peritubal area. That portion of the posterior canal wall and annulus that overlies the deep recess of the tympanum posterior to the round window and stapes must be removed, so that the processus pyramidalis is in view. With these impeding margins of the annulus out of the way, the entire medial wall of the tympanum is exposed; only negligence and timidity can account for failure to complete the tympanic surgery.

The facial ridge must be lowered to the extreme limit—whatever the size of the mastoid—so that when the mastoid cavity is viewed postoperatively through a large speculum one can look directly into the area posterior to the facial ridge.

In many patients with chronic suppurative otitis one may find considerable cellular development in the perilabyrinthine area. It

is extremely important, particularly in the vascular type of bone, to expose thoroughly the semicircular canals. Any leads in the bone surrounding the canals must be followed. This step is particularly important where there is danger of overlooking cholesteatomatous prolongations that have penetrated into these spaces.

The tegmen should be level and smooth.

The removal of the tensor tympani muscle, particularly where disease is found in the region of the tube, is indicated. If the mucosa of the middle ear and the tube are not diseased, the muscle need not be removed.

Facial Nerve. As for the facial nerve, the danger always exists, of course, that it may be injured. But in the hands of an expert surgeon, it is a rare accident in this day of good illumination and magnification. What is important, as Lempert has pointed out, is that in the event of injury there is no excuse for not knowing exactly where and when the damage was done or for not correcting it on the spot.

Cavity. Our operation is completed; the bone work, finished. What are we going to do with the cavity? A small cavity has the advantage of requiring a minimal amount of epithelialization, minimizing the accumulation of desquamated epithelium. But a small cavity cannot always be our happy lot. When we are faced with an extensively diseased mastoid, or one with pre-existing pneumatization, where disease or cholesteatoma has penetrated into all the cell spaces, we are going to have to solve the problem of a large cavity.

Since mastoid surgery began, surgeons have struggled with cavity obliteration—muscle graft, periosteal graft—and sometimes they have done it successfully. I am in favor of these grafts if they are not allowed to extend into the epitympanum and middle ear space.

What about leaving the cavity as is and letting nature cover it with a granulating surface which would ultimately epithelize? Or should we graft?—immediately or later?

I prefer to use the primary graft, not the least of whose virtues is that it shortens the healing period. I like a thin graft and place five or six small sections in the cavity, fitted much like wallpaper to the bone surfaces and with one or two small incisions in each graft to allow serum to escape from between the bone and the graft. To retain the grafts in position one can use small pellets or squares of Gelfoam moistened in thrombin and then squeezed almost dry. Placed over the Gelfoam to exert a continuous pressure are small balls of paraffin mesh. Gelfoam is so pliable that at the slightest pressure it ex-

pands into recesses and, without dislocating them, holds the grafts firmly against the bone. After five or six days, this packing is painlessly removed by suction. Should an odor be detected after a few days, signaling infection, the paraffin mesh is removed but the Gelfoam underneath it, if not macerated, is left in place and the surfaces dusted with one per cent Polymixin to one gram Biosorb, or, after culturing secretions, use the antibiotic powder to which the organism shows greatest sensitivity. The immediate after treatment following removal of the Gelfoam is a simple matter of gently absorbing secretions with cotton applicators and dusting the surfaces with the antibiotic powder of choice.

SUMMARY

I have already said that the basic principles of radical mastoid surgery were clarified in the last century. The fundamental rules of actual surgical procedure, the detailed steps by which one accomplishes the objectives of the operation, were laid down for us many years ago. I do not pretend to offer anything new in the way of principle or precept. I offer no innovation, either in concept or performance.

My purpose, rather, has been to underline, as emphatically as possible the duty the surgeon owes his patient to do everything within his power, under the conditions of the given case, to conserve or improve what hearing is present. To fulfill this obligation, the surgeon must be ready to observe a certain flexibility in applying the classic principles and the ground-rules of radical mastoidectomy. He must be prepared to depart from that concept and those rules if by so doing he can safely conserve the hearing. He must not start out with a fixed idea of the complete radical, but look upon each operation as a "custom job," if you like, cut to fill the specific requirements of each individual case.

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THE SIGNIFICANCE OF THE ARTERIOVENOUS ARCADES
OF THE SPIRAL LIGAMENT OF THE COCHLEA

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There are at present two major methods of studying the small blood vessels of the spiral ligament: 1) By injection techniques in dead animals, with various chemical treatments of the bony cochlea to permit serial sectioning or microscopic dissection of its blood supply; 2) fenestration of the cochlea over the spiral ligament in living guinea pigs with so little trauma that an approximation of normal blood flow is retained.[1]

Either approach offers technical difficulties of considerable magnitude. It is the opinion of our group[1] that the latter method has unlimited possibilities for advancing basic physiological knowledge of the inner ear. By properly designed experiments much light may also be thrown upon the pathological physiology of the organ of hearing.

It is because the arteriovenous arcades of the spiral ligament fascinated Dr. H. P. Mosher, in whose honor this article is published, that this subject is discussed.

WHAT IS AN ARTERIOVENOUS ARCADE?

Since the clinician dealing in the practicalities of aural disease cannot be expected to have an expert knowledge of small blood vessels comparable to that of a physiologist making a career of such work, a brief summary of a few important observations in this field can be helpful at this point.

1. The capillary bed is a vast network of branching and anastomosing cylinders. Thus Krogh[2] has pointed out that if one supposes a man's muscles to weigh 50 kg, and his capillaries in this tissue to number 2000 per sq mm, the total length of all these tubes together would encircle the globe about two and a half times, and have a total surface of 6,300 square meters!

2.   The arteriole is a cone-shaped vessel having linear blood flow going from the larger to the smaller end.   This fact permits its recognition easily in living animal experiments, but not in dead ears. It subdivides into capillaries; but see No. 4 below.

3.   The venule receives capillary flow, and flow from other venules.   It is cone-shaped with linear flow from the smaller to the larger end.

4.   The arteriovenous arcade is an arteriole carrying arterial blood directly to a collecting venule, without intermediation of the capillary bed.   In 1895 Stewart[3] calculated that 1 cu mm of blood required six hours to pass through an average-sized capillary at a rate of 0.5 mm per second.   But the average arteriovenous shunt seen by us in the spiral ligament is capable of transferring this amount of blood in a matter of seconds.   The arcades are wider than capillaries and thicker walled.   Krogh[2] remarks "The blood passing through them will probably fail to give off the normal amount of oxygen and other substances which it is supposed to carry to the tissues, because it is not exposed to the thin walls and large surface of the capillaries, but for the dissipation of heat the surface available in vessels of less than 0.1 mm diameter is ample."   It is likely that a major function of these vessels is heat transfer to the tissue.

5.   Capillary pressure relationships assist in visualizing clearly the physiology of small vessels.   Harper[4] approximates the figures as follows:

ARTERIAL SIDE OF CAPILLARY LOOP

Blood hydrostatic pressure	>	30 mm Hg
Tissue hydrostatic pressure	<	8 mm Hg
Net	>	22 mm Hg
Protein osmotic pressure inside vessel	<	25 mm Hg
Protein osmotic pressure outside vessel	>	10 mm Hg
Net	<	15 mm Hg

Subtracting pressure gradients into and out of the vessel, as indicated by the arrows: 22 minus 15 = 7 mm Hg > as net filtration pressure. pressure.

VENOUS SIDE OF CAPILLARY LOOP

Blood hydrostatic pressure	>	15 mm Hg
Tissue hydrostatic pressure	<	8 mm Hg
Net	>	7 mm Hg
Protein osmotic pressure inside vessel	<	25 mm Hg
Protein osmotic pressure outside vessel	>	10 mm Hg
Net	<	15 mm Hg

Subtracting pressure gradients into and out of the vessel, as indicated by the arrows: 15 minus 7 = 8 mm Hg < absorption pressure.

Landis[5] describes the single capillary as "The smallest unit of interchange between blood and tissue," with a wide range of normal variation. Thus, "single capillaries differ in diameter, length, and the nature of their connections with the smallest arterioles and venules." The wall consists of a single layer of thin endothelial cells placed edge to edge and bridged by an intercellular substance. This mosaic-type membrane is about one micron in thickness.

In contrast, an arteriovenous arcade roughly duplicates the structure of an arteriole of the same size up to the point of its joining a collecting venule. This permits heat dissipation but is not adequate as is the capillary for the diffusion, filtration and reabsorption required in tissue nutrition. It is to be remembered that the capillary beds of body surfaces are of prime importance in heat regulation because of their extent and capacity to receive an enormous volume of blood from internal organs, where the temperature is 2° F or 3° F higher. But the arcades are capable of a very rapid delivery of heat to a given area.

OBSERVATIONS

Our group[1] has described the exposure of the spiral ligament and stria vascularis in living anesthetized guinea pigs. By such means it has been possible to observe microscopically the living circulation of this area, in contrast to the array of histological post-mortem ear techniques used by other investigators. Arterioles, capillaries, venules and arteriovenous arcades as previously defined were seen. Their arrangement as viewed by this method may be summarized as follows:

1. The arterioles enter the field from the scala vestibuli side and supply an internal group of branches to the stria vascularis capillaries, and an external group to the capillaries of the remainder of the aspiral ligament.

2.   A third group of arterioles, the arteriovenous arcades, at least as numerous as each of the above ones, ends in the transverse or vertical collecting venules.

3.   The transverse collecting venule lies in a spiral plane about parallel to the external spiral sulcus of the cochlear duct, but well external to it (from the modiolar axis of the cochlea).   This vein measures about 40 microns in diameter, and parallels the pigment cell distribution of the spiral ligament.   It receives capillaries from both networks, and picks up venular tributaries.   Its morphology and size are against its being itself a capillary.   The vertical collecting venules leave the field by going over the scala tympani, and receive blood from the transverse collecting venule itself, and independently from the other tributaries of this venule.

4.   In well-prepared experimental fields it is possible to identify the line of attachment of Reissner's membrane.   Here can be seen one set of arterioles sinking into the pigment cell area to break up into the capillaries of the stria vascularis, and a second set lying nearer the endosteum to supply the remainder of the capillaries which we have observed.

It is evident, then, that both capillary beds (and any others not visible by our method) can be by-passed by the arteriovenous shunts.

PHYSIOLOGICAL SIGNIFICANCE OF THE ARTERIOVENOUS ARCADES

1.   It would be logical to assume, as did Agazzi[6] that the arteriovenous arcades act to by-pass blood upon occasion away from capillary networks.   Although it is true that individual arterioles (and venules) appeared normally to dilate and contract independently, *this was not true in en masse reactions*.   In general, when arterioles were thrown into contraction, as in anaphylactic shock[7] the arteriovenous arcade contracted also.   It has not as yet been possible to devise any experiment for inducing in the living guinea pig mass contraction of the arteriovenous anastomoses with simultaneous dilatation of the arterioles to the capillary beds, or vice versa.   Agazzi constructed models to demonstrate this hypothetical physiological state.   His purpose was to implement his view that these arcades are utilized for the regulation of blood flow through the stria vascularis in accord with the need for the regulation of secretion of endolymph. That is, when less blood was needed in the capillaries of the stria vascularis, he supposed it to be quickly by-passed away from the field by the shunts; and vice versa.

It is true experimentally that the speed of contracture of the spiral ligament arteriovenous arcades in anaphylactic shock is slower

than that of the arterioles to the capillary beds, provided the guinea pig does not die quickly, or recovers. And if the animal dies, flow ceases last in the arcades. But there is no gross reciprocal relationship in anaphylactic shock (Zinsser[8] presumed that allergy in man is based on an immunological mechanism fundamentally identical with anaphylaxis in animals) whereby arterioles to the capillary beds of the spiral ligament contract while the arteriovenous arcades fail to do so. This fact does not rule out Agazzi's unproven hypothesis, since in the normal experimental fenestration field we have found that individual arterioles and arteriovenous arcades contract and dilate independently. This is in line with observations of these structures in other tissues; thus, Clark and Clark[9] found in the rabbit ear chamber an enormous range of variation in the character, rate and duration of the spontaneous contractions of the arteriovenous anastomoses.

In the normal control of the circulation of the spiral ligament such spontaneous contractions and dilatations must be physiologically important.

2. In a satisfactory fenestration field over the guinea pig spiral ligament, blood flow is so "smooth" that intravascular elements are poorly defined at moderate magnifications. In the presence of trauma, histamine or anaphylactic shock, dramatic changes appear. Thus, in the last condition red cell aggregates appear, masses highly refractile to light ("white emboli") are seen, and either type of mass may block an arcade, arteriole, or venule as a thrombus. If the animal survived, these thrombi did not disappear over an observation period of up to four hours, but otherwise the circulation reverted to normal. The implication is that such thrombi prevented the involved vessels from performing their normal function at least for a substantial length of time, with the likelihood of tissue damage resulting.

3. Function in Heat Regulation: An exhaustive search of the literature has revealed that arteriovenous arcades are found in many tissues.[10-34] It is generally agreed that these vessels when dilated can bring an enormous amount of body heat to a tissue, especially a peripheral one such as the fingers or eyelids. Clark[35] estimates that an arteriovenous arcade with an internal diameter of 40 microns will pass better than 256 times as much blood as a capillary of 10 microns diameter (in accordance with the law that volume flow varies with the fourth power of the aperture measurement). Since the speed of the flow must be considered as well as the diameter of the vessel, the mechanical advantage for the transfer of warm arterial blood to the venous side through an arcade is obvious in dispersing heat from blood to a tissue. Physiologists believe, for example, that the rich

supply of arteriovenous shunts in the eyelids prevent their freezing when exposed to low temperatures.

A hint as to the basic function of arteriovenous arcades in heat distribution is Grosser's[36] observation that these vessels are not to be found in the bodies of snakes and other cold-blooded animals. (An exception is the frog.[37])

The ease of transmission of heat or cold from the external environment to the inner ear is known to all otologists in their use of caloric tests to determine labyrinthine function. Since the arteriovenous arcades appear to provide a superb physiological heat regulating system for the cochlea, it is desirable to investigate their presence in the vestibular labyrinth. It is known clinically that prolonged exposure of an ear to cold wind may cause temporary deafness; this might be due to overwhelming the inner ear radiator mechanism just described. If the same type of heat regulation is present in the vestibular organs, caloric responses could be a result of merely altering normal tonus relationships.

The cochlea of the guinea pig is surrounded by the air space of the bulla. The spiral ligament is therefore separated from air only by a thin layer of bone. In the human ear, a considerable segment of the cochlear bony wall lies against the air space of the middle ear. This arrangement provides for ease of radiation of excess heat from the inner ear to the middle ear air space. This may, therefore, aid not only in regulation of the cochlear but also the tympanic cavity temperature level. Attempts by us to measure the temperature of the intact guinea pig bulla air space by use of a thermocouple have thus far been unsuccessful.

4. In normal vascular relationships in man, clinicians are sometimes unaware of a fact very clearly realized by physiologists; namely, that the blood volume is completely inadequate to fill all of the capillary beds at the same time. Yet the far less numerous arterioles and venules are filled, but constantly fluctuating in internal diameter by spontaneous contractions and dilatations.

If, for example, in a resting muscle only 10 per cent of the total capillaries are open and functioning, and in exercise the oxygen requirement of the tissue demands that 90 per cent be open, blood can be quickly shifted from the splenchnic area (holding about one-third of total blood volume) to the muscle in question. A simile is the shifting of a division of troops in an army from one front to another, with inability and lack of necessity to crowd masses of soldiers onto all fronts at the same time!

Therefore it is possible that the arteriovenous arcades, wherever found, whether in the ear or elsewhere, function in part to mobilize blood to an area of greater usefulness. The independent contractility of individual arterioles, venules, and arteriovenous arcades may be important as an exact reflection of the blood flow required for nutrition of a given tissue segment at a particular time instant in the capillary bed area.

5. But the arteriovenous arcades, closely resembling arterioles of like size, do not have a direct function in tissue nutrition; they are not capillary competitors!

CONCLUSION

Recent experimental work in studying the circulation of the spiral ligament in living guinea pigs has made the arteriovenous arcades of interest to practical clinicians as well as to those who are basic scientists.

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REFERENCES

1. Weille, F. L., Gargano, S. R., Pfister, R., Martinez, D. and Irwin, J. W.: Circulation of the Spiral Ligament and Stria Vascularis of Living Guinea Pig, Arch. Otolaryng. 59:731 (June) 1954.

2. Krogh, A.: The Anatomy and Physiology of Capillaries, rev. ed., New Haven, Yale Univ. Press, p. 31, 1936.

3. Stewart, G. N.: Manual of Physiology, London, Baillière, Tindall and Cox, cited by Krogh[2] p. 104, 1895.

4. Harper, H. A.: Review of Physiological Chemistry, ed. 3, Palo Alto, Univ. Med. Pub., 1951.

5. Landis, E. M.: Capillary Permeability and the Factors Affecting the Composition of Capillary Filtrate, Ann. N. Y. Acad. of Sci. 46:713 (Sept.) 1946.

6. Agazzi, C.: Appunti di idrodinarmica vascolare del legamento spirale, Arch. ital. di otol. 60:40 (1-2) 1949.

7. Weille, F. L., Martinez, D. E., Gargano, S. R., Irwin, J. W., Gilchrist, M. and Gordon, P.: An Experimental Study of the Small Vessels of the Spiral Ligament and Stria Vascularis of Living Guinea Pig During Anaphylaxis, Laryngoscope 64: 656 (Aug.) 1954.

8. Zinsser, Hans: Personal Communication.

9. Clark, E. R. and Clark, E. L.: Observations on Living Arteriovenous Anastomoses As Seen in Transparent Chambers Introduced Into the Rabbit's Ear, Am. J. Anat. 54:229, 1934.

10. Benninghoff, A.: Die erste Entstehung der Gefässe in des Herzens: N. Arterio-venöses Anastomosen, Glomus Coccygium und Polstarterien, in Möllendorf's Handb. d. mikr. Anat. des Menschen 6:1, 107, 1930.

11. Clara, M.: Die arterio-venösen Anastomosen der Vögel und Säugetiere, Ztschr. f. d. ges. Anat. (Abt 3) 27:246 (Nov.) 1927.

12. Bauer, W., Dale, H. H., Poulsson, L. T. and Richards, D. W.: The Control of Circulation Through the Liver, J. Physiol. 74:343 (Apr.) 1932.

13. Knisely, M. H., Bloch, E. H. and Warner, L.: Selective Phagocytosis: I. Microscopic Observations Concerning the Regulation of the Blood Flow Through the Liver and Other Organs and the Mechanism and Rate of Phagocytic Removal of Particles from the Blood, Det. Kongelige Danske Vdnsk. Slsk. Biol. Skr. 4:7, 1948.

14. Trueta, J., Barclay, E. A., Daniel, P. N., Franklin, K. J. and Prichard, M. M. L.: Studies of the Renal Circulation, Springfield, Ill., Charles C. Thomas, 1947.

15. Goodwin, W. E., Sloan, R. D. and Scott, W. W.: The "Trueta" Renal Vascular "Shunt;" an Experimental Demonstration of Neurovascular Control of the Renal Circulation in the Rabbit, Cat, Dog, and Monkey, J. Urol. 61:1010 (June) 1949.

16. Knisely, M. H.: Spleen Studies: I. Microscopic Observations of the Circulatory System of Living Unstimulated Mammalian Spleens, Anat. Rec. 65:23 (Apr.) 1936; II. Microscopic Observations of the Circulatory System of Living Traumatized Spleens, and of Dying Spleens, ibid. 65:131 (May) 1936.

17. Peck, H. M. and Hoerr, N. L.: The Intermediary Circulation in the Red Pulp of the Mouse Spleen, Anat. Rec. 109:447 (Mar.) 1951.

18. Prinzmetal, M., Simkin, B., Bergman, H. C. and Kruger, H. E.: Studies on the Coronary Circulation; I. The Collateral Circulation of the Normal Human Heart by Coronary Perfusion with Radioactive Erythrocytes and Glass Spheres, Am. Heart J. 33:420 (Apr.) 1947.

19. Prinzmetal, M., Ornitz, E. M., Simkin, B. and Bergman, H. C.: Arteriovenous Anastomoses in Liver, Spleen, and Lungs, Am. J. Physiol. 152:48 (Jan.) 1948.

20. Tobin, C. E. and Zariguiey, M. O.: Arteriovenous Shunts in the Human Lung, Proc. Soc. Exper. Biol. & Med. 75:827 (Dec.) 1950.

21. Knisely, H. M.: Microscopic Observations on Circulatory System of Living Transilluminated Mammalian Spleens and Parturient Uteri, Proc. Soc. Exper. Biol. & Med. 32:212 (Oct.) 1934.

22. Danesino, V.: Dispositivi di blocco ed. anastomosi arterio-venose nei vasi fetali, della placenta umana, Arch. di ostet. e ginec. 55:251 (3) 1950.

23. Marley, A. and Soldati, M.: Contributo all' innervazione dell' ovario: innervazione delle anastomosi arterio-venose, Boll. Soc. ital. Biol. sper. 27:640 (4) 1951.

24. Hyrtl, J.: Anatomical Notes, Natural History Review, London, 1862.

25. Hoyer, H.: Ueber unmittelbara Einmündung kleinster Arterien in Gefäss-äste venösen Charakters, Arch. f. mikr. Anat. 13:603, 1877.

26. Grant, R. T. and Bland, E. F.: Observations on Arteriovenous Anastomoses in Human Skin and in the Bird's Foot with Special Reference to the Reaction to Cold, Heart 15:385 (July) 1931.

27. Schumacher, S. von: Orterio-venösen Anastomosen in den Zehen der Vögel, Arch. f. mikr. Anat. 87:309 (Oct.) 1915.

28. Arnold, J.: Ueber die Glomeruli caudales der Säugethiere, Virchow's Arch. (Arch. f. path. Anat.) 39:497 (4) 1867.

29. Lewis, T.: Observations Upon the Reactions of the Vessels of the Human Skin to Cold, Heart 15:177 (May) 1930.

30. Grosser, O.: Ueber arterio-venösen Anastomosen an den Extremitätenden beim Menschen und krallentragenden Säugethiere, Arch. f. mikr. Anat. 60:191, 1902.

31. Deysach, L. J.: The Nature and Location of the "Sphincter Mechanism" in the Liver as Determined by Drug Actions and Vascular Injections, Am. J. Physiol. 132:713 (Apr.) 1941.

32. Grant, R. T. and Bland, E. F.[24]

33. Gasparini, F.: Disposizione Anastomotiche artero-venose nel Palato Molle, Boll. Soc. ital. Biol. sper. 23:921 (8) 1947.

34. Staubesand, J.: Anat. Anz. 97: (suppl. on Verh. anat. Ges.) 68, 1950.

35. Clark, E. R.: Arterio-Venous Anastomoses, Physiol. Rev. 18:229 (Apr.) 1938.

36. Grosser, O.: Bau und Function der arterio-venösen Anastomosen, besonders bei den Chiropteren, Wien, klin. Wschr. 14:623 (May) 1901.

37. Irwin, John W.: personal communication, 1955.

XXI

DANGERS INHERENT IN THE NONSURGICAL CONCEPT OF ACUTE SUPPURATIVE OTITIS MEDIA

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The purpose of this paper is to call attention to some of the problems which have recently arisen in the treatment of acute otitis media, in the ear department of the Massachusetts Eye and Ear Infirmary. In no sense is this a statistical study. Rather it is the intent of the authors to point out certain errors in otologic thinking based on an analysis of case histories.

The authors would like to review some of the trends in treatment of otitis media since the start of drug therapy as a background to the subject under discussion. When antibiotics and chemotherapeutics were first introduced there was, as we all remember, an extraordinary drop in the incidence of otitis media and especially of the complication which used to follow this infection. It seemed at that time the millennium was at hand when medical treatment with an antibiotic would completely supplant the surgical approach to the disease. Those who remembered fluctutations in the incidence of otitis media were somewhat skeptical and felt that there was still a possibility that the sudden drop in otitis media might be a cyclic one; that sooner or later the virulent organisms would reappear in more epidemic form and then we would have an increase in the severity and incidence of the disease. However, as time went on, this did not occur and generally the medical world felt that indeed we had eliminated otitis media and mastoiditis or at least the later.

Then certain disturbing factors appeared. Strains of bacteria resistant to penicillin or to some of the later drugs were discovered. Where the dosage originally recommended for a certain strain of bacteria had been adequate, as time went on, larger doses and more prolonged treatment were required to accomplish the same result. In addition to this, certain patients developed allergic reactions. This was a real problem, for many people had been sensitized during the

early years by the use of antibiotic drugs, chiefly penicillin, with the result that these individuals could never again be given this treatment. One wondered then what would happen if such a person were stricken with a really dangerous respiratory infection and could not be given the benefit of penicillin. Then, too, at the beginning of this era, it was not well established whether one should stop treatment at the moment symptoms appeared to abate or continue the treatment for a longer period of time. This was further complicated by claims that some diseases could be treated with a one-shot dose. It was not uncommon for a patient to come to one's office who had been given a few courses of an antibiotic and then when the symptoms failed to improve shifted to another antibiotic and so on. No real scientific approach was made in such cases. As it was rare for medical students to observe the clinical course of otitis media and mastoiditis, many graduated with the feeling that these conditions were of no consequence or no longer existed or if they did exist, a short course of an antibiotic was all that was needed. Practically a whole generation has grown up in this delusion. This does not mean that real efforts were not made to establish antibiotic treatment on a sound scientific basis but the knowledge gained from these studies has not yet been propagated throughout the profession.

While the authors do not intend to recapitulate the treatment of otitis media, it seems appropriate to underscore some of the cardinal principles as they appear to us in the light of our recent experience. In the first place, an accurate bacteriological evaluation should be undertaken at the very beginning. This should be done before any antibiotic or chemotherapeutic drug has been given as, otherwise, the organism though present, may fail to grow in vitro. If there are signs of secretion in the middle ear, this can best be accomplished by a myringotomy and then by taking a culture directly from the escaping fluid. If the drum and middle ear do not appear to have reached the secretory or purulent stage, a culture from the nasopharynx, especially from the region of the eustachian tube, may be of great help, although in certain cases this is not as accurate as a culture taken directly from the middle ear secretions. This culture should be planted in a meat broth tube and also planted on blood agar. Even in cases which have already been given an antibiotic, it may not be too late to obtain important information especially in dealing with a resistent strain. Many ways are recommended for testing the sensitivity of the organism to a given antibiotic and these tests should be carried out where it is a question of dealing with a resistent strain.

As many patients have had previous attacks of otitis media, apparently "cured" by previous administration of drugs, it would be well

to have x-rays taken of these cases to determine the extent of bone changes which have occurred and it may well be that cholesteatoma has begun to form even where there has been apparently a good recovery from these previous attacks. Having provided drainage from the middle ear and undertaken treatment with the proper antibiotic agent, it is extremely important to follow the course of the disease not only by clinical observation but by x-ray. It is our opinion that this treatment should be continued until there is no further discharge, until all landmarks have returned and there is no evidence of mastoid involvement.

In the case of children who have repeated attacks of otitis media, a therapeutic adenoidectomy is recommended. Where tubal infection reappears, attention should be paid to the possibility of residual infection in the paranasal sinuses. Finally, the authors would like to reaffirm that otitis media is still a surgical disease. It may not and obviously does not mean in every case that the patient is headed for a mastoidectomy. But the mental attitude of the attending physician should be that this is primarily a surgical problem; that the principles of surgical drainage even where antibiotic treatment is effective should be insisted upon. This has been objected to on occasion by pediatricians who claim that early myringotomy did not always obviate the necessity of mastoidectomy. Consequently, by reasoning backward, mastoiditis was not prevented by myringotomy; that "nature" either would clear up the infection or in spite of human interference, the patient would develop mastoiditis. This fatalistic attitude is still held in some quarters. The cases we report here, point up the following errors or pitfalls which we think should be emphasized: 1) A mistaken idea that mostoiditis has disappeared from the medical picture; that an attack of otitis media carries with it no dangers and requires only a short course of antibiotic treatment to reduce the temperature and pain; 2) the conception that this is a medical disease and that there is no need for myringotomy and that there is no need for myringotomy and that there is no possibility of surgical complications provided antibiotics are given in large doses; 3) treatment with antibiotics without previous determination of the bacteria involved; 4) a reluctance to call for a consultation from an otologist until obvious signs of mastoiditis have presented themselves; a tendency for the pediatrician to take over the early stages of an otitis media without consultation.

In order to evaluate the present day incidence of surgical mastoiditis in cases of acute otitis media, one has to go back to the pre-chemotherapeutic days for a basis of comparison. A review of literature of that period shows a difference in percentage of cases requiring

operation in the experience of different observers. This may be
evidence of epidemic or cyclic factors as Tobey[1] pointed out. In 1946
House[2] reported 1241 patients not treated with sulphonamids, of
whom whom 45.9 per cent required mastoidectomy. Also some sta-
tistics compare mastoidectomy cases with "otitis media and mastoid-
itis" and some with "otitis media" alone. In 1934 Page[3] reported
that only 6 per cent of 300 cases of otitis media required mastoid-
ectomy. In these cases myringotomy has been performed. In 1942
Richardson[4] reviewing 625 cases of acute otitis media in which my-
ringotomy had been done, found that without chemotherapy the
incidence of mastoidectomy was only 5.3 per cent. Butler[5] in 1948
found that out of 461 cases of purulent otitis media treated without
chemotherapy, only two simple mastoidectomies were done. Granting
that the indications for surgical intervention may vary in different
regions, still it would seem that barring an epidemic, the incidence
of surgical mastoiditis in acute suppurative otitis media is 6 per cent
or less under normal conditions with myringotomy and without
chemotherapy. One must also recognize that many cases of acute
otitis media in certain communities never reach the hospital; there-
fore, it is difficult to find a figure for the over-all incidence of acute
otitis media in those communities. Where patients with acute otitis
media are admitted directly to the hospital, of course, one has a base
line on which to determine the percentage of cures or successes with
any form of treatment. Evans[6] in a series of cases of acute otitis
media admitted to the out-patient department of the Massachusetts
Eye and Ear Infirmary over a four-month period in 1948 found that
out of 45 cases seen in that department and treated by early myrin-
gotomy all recovered without mastoidectomy. Some of these were
given antibiotics and some were not. During the same period 30
cases were admitted directly to the hospital from other sources than
the out-patient department for acute surgical mastoiditis. In none
of these cases of surgical mastoiditis had an early myringotomy been
performed. They had all been given some form of chemotherapy or
antibiotic treatment. It was difficult to determine in what dosage
or for how long a period or how early in the disease this treatment had
been instituted. But the important point was that in none of them
had a myringotomy been considered. Zonderman[7] in a discussion of
the use and abuse of antibiotics and the importance of myringotomy,
comes to the conclusion that the most common error in the therapy
of otitis media is treatment with antibiotic without myringotomy.
He points out that this may lead to a temporary suppression of the
bacterial infection with subsidence of the acute symptoms of otalgia
and fever followed shortly by a more severe and hazardous recur-
rence. He also points out that without myringotomy and complete

eradication of the original infection, there is great danger that the patient may develop a chronic otitis with loss of hearing function. Davison[8] believes that the chief reasons for failure of treatment in acute suppurative otitis media are: 1) inadequate dosage, 2) deficiency of antibacterial antibody, 3) a focus of undrained pus and, 4) intravascular infection such as thrombophlebitis. In his series from the years 1937 to 1953 he says that the incidence of surgical mastoiditis has dropped from 58.9 per cent to 0 per cent due to early myringotomy and the new sulfa and antibiotic drugs, while the cases of acute otitis media have dropped only from 202 to 122. In other words, although the disease of otitis media has not been eradicated, he concludes that mastoiditis can and should be prevented by early myringotomy and by early administration of one of the antibiotics, preferably penicillin in adequate doses. To determine what the proper dose should be, he quotes Fleming[9] as follows: "(1) It should be used only when there is an infection by a penicillin sensitive microbe. (2) Penicillin must be administered in such a way that it comes in contact with the infecting microbe. (3) The dose should be such that in the infected area concentration of penicillin is sufficient to destroy the bacteria. (4) The treatment should be persisted in until the infection is defeated." Davison further states "in order to diminish the tendency for emergence of resistant strains it is desirable to prescribe from the outset a dosage sufficient to insure maintenance of an optimum concentration of the antibiotic in the body fluids until clinical symptoms of the infection subside." After the symptoms have subsided, it is advisable to continue the use of the antibiotic for a period of 48 hours and discontinue its use without a tapering off period.

These studies suggest the following conclusions. In the days before chemotherapy surgical mastoiditis could be given a percentage relationship to otitis media. It is very difficult to do so now, as we have pointed out, since relatively few cases of otitis media now reach the clinics where a statistical estimate can be made. Should we now apply the six per cent relationship, it would mean that in our clinic fifteen simple mastoidectomies done on the present service represent an incidence of 250 cases of otitis media. This may well be inaccurate on the low side as many cases of acute upper respiratory infections may be stopped short of acute otitis media by the general use of the antibiotics, so that we are seeing in the operating room only the neglected, inadequately treated or drug resistant disease. But although the number of mastoidectomies is much less than in the earliest days, the assumption is that as we still have cases of otitis media, and the dangers of mastoid involvement must be reckoned with.

In the recent months on the otological service of the Massachu-
setts Eye and Ear Infirmary, 15 cases came to operation which em-
phasize the need for reaffirming the principles of treatment as out-
lined above.   Six of these histories are given here with comments.

REPORTS OF CASES

CASE 1

A girl, aged three months. Two months prior to admission to the ear service
she was given nose drops for a head cold. A short time afterwards the mother
noted a "milky" fluid in the left external auditory canal. She stated that she
swabbed this material frequently from the canal. Two weeks prior to admission,
she was again given nose drops. As the mother felt that the baby was in good
health, although she continued to notice an aural discharge, she did not report to
the outpatient department until the present admission. About fourteen hours prior
to admission the baby began to refuse her feeding and vomited once. She felt hot
and the mother observed purulent discharge from her right ear also. On examina-
tion the patient appeared pale and ill and cried vigorously. The skin was hot but
not dehydrated. The head was normal in appearance and the anterior fontanel was
soft. Examination of the eyes was negative. Ear examination revealed that the
upper part of the left drum was red and edematous with obscured landmarks. There
was a small anterior inferior perforation from which fluid appeared to be flowing.
The right drum appeared red. There was a good deal of mucoid nasal discharge.
Her pharynx was mildly injected. Her neck was supple. There were many en-
larged posterior auricular and anterior cervical lymph nodes. Her chest was clear
and the cardiovascular system appeared to be normal. The neurological exam-
ination was negative. The white blood count was 10,500 cells with a differential of
67% polymorphonuclears, 29% lymphocytes, 4% monocytes. There were normal
platelets and a slight hypochromia of the red blood corpuscles. Hemoglobin 10.9
grms per cent. The patient's admission temperature was 102° by rectum. On the
day following admission her temperature rose to 105.° A myringotomy of the
left drum was done on this day. Cultures of the purulent exudate from the left
ear showed staphylococcus aureus and hemophilus influenzae. The patient was
given aureomycin, 125 mg every eight hours. She continued to be febrile with
peaks up to 103° in spite of the fact that the sensitivity of the organisms growing
out of the discharge was reported to be satisfactory to aureomycin. An x-ray of
the mastoids, three days after admission, revealed that there was a loss of cell wall
definition and considerable destruction of the left mastoid cells. A blood culture
taken on the night of admission showed h. influenzae. Four days later it was
noted that the infant had a bulging fontanel. A lumbar puncture performed at
that time revealed cloudy cerebrospinal fluid with 1585 cells, 68% polymorpho-
nuclears, 32% mononuclears, with 68 mgs per cent sugar and 62 mgs per cent
protein. The child was placed on penicillin, 6000 units a day, chloromycetin, 200
mgs a day, and sulfadiazine, 200 mgs per day. At this point the otological service
was asked to take the child over for surgery. A left simple mastoidectomy was
performed. It was noted that the mastoid cells were broken down and filled with
pus. The dura and lateral sinus were uncovered and appeared to be uninvolved.
A culture of the spinal fluid taken from a lumbar puncture on the day of the
operation revealed type B, H. influenzae. The child continued to show meningeal
irritation so it was the opinion of the otological service that probably the right
mastoid also had been involved as it had been noted on admission that the drum
was red and the mother had noticed a discharge two weeks before. Therefore, a
right simple mastoidectomy was done. The mastoid was not decalcified but it was
hemorrhagic. Two days later the child was afebrile and the signs of meningeal
disease disappeared by the fourth postoperative day. Twelve days after the first

operation, as the child had remained afebrile and the ears were dry and the wounds healed, all antibiotic therapy was stopped. On the sixteenth postoperative day a lumbar puncture was performed with negative findings.

Comment: In this case it would seem that not enough attention was paid to the mother's statement that the child had a running ear and that an otologist was not called to see the child until complications had already occurred, approximately two months subsequent to the first symptoms. Myringotomy was not done at the onset nor was any bacterial analysis, proper chemotherapy or antibiotic treatment started early in the disease. Consequently, when the child was finally admitted, the x-ray showed destruction of one mastoid, and there were already signs of meningeal involvement. Once a myringotomy and bacterial studies of the discharge had been obtained, antibiotic treatment was started on adequate doses. Of course, by this time it was too late to prevent a surgical mastoid.

CASE 2

A girl, aged 14, was admitted on November 30, 1954, for recurrent otorrhea and pain in the left ear. Two years before this she had spontaneous ontset of otorrhea and pain in her left ear. Treatment at that time consisted of sulfadiazine and ear drops. The otorrhea and pain subsided in two weeks and the patient remained symptom free for five or six months. Then she had a second episode of otorrhea and pain in the left ear, was treated in the same manner, and recovered in ten days. The patient was first seen in our outpatient department in September of 1953. She complained of pain and otorrhea in the left ear for the third time and was treated with oral penicillin and chloromycetin powder in her ear. Again the pain and discharge ceased after ten days of therapy. The otorrhea and pain in the left ear returned again for the fourth time in January of 1954. This time she was treated with intramuscular penicillin and the discharge ceased after two weeks of therapy. Examination of the ear six months later was negative. In the summer of 1954, the patient did considerable swimming, following which she noted a discharge from her left ear without pain. Two weeks before admission this otorrhea increased and she complained of earache. Four days before her admission, she had vertigo of a few minutes' duration. This was repeated on the day of her admission. She had no nausea or vomiting, tinnitus or headache. X-rays of the sinuses and chest were negative. Lumbar puncture was within normal limits. Hematology and urinalysis were normal. The hearing was normal. X-ray of mastoids showed: the right mastoid normal; left mastoid sclerosis with no destruction. A culture from the left ear showed B. pyrocyaneus. The patient was placed on conservative therapy, consisting of bacitracin, streptomycin, and polymixin ear drops, and chloromycetin by mouth. The otorrhea continued. Because of failure of conservative therapy and persistence of symptoms, a left simple mastoidectomy was performed on December 7, 1954, one week after admission. The mastoid cells were found to be small and the partitions, though present, were softened. The mastoid healed gradually and the middle ear resolved slowly postoperatively.

Comment: In this case the treatment had been inadequate on previous occasions. Ear drops, occasional treatment with penicillin, and sulfa drugs apparently had caused each attack to subside after about ten days of treatment but by the time she arrived at the hos-

pital, on this admission, her mastoid had already begun to show sclerosis. The bacteriology was bacillus pyocyaneus, an organism resistant to the usual antibiotics. Apparently in her previous attacks she had not had a myringotomy nor had she had a satisfactory bacteriological determination. Too much reliance was placed on medication which probably was in too small and ineffective doses. The danger of such treatment is the tendency to chronicity with extension of disease to surrounding areas and the emergence of organisms resistant to the usual drugs.

CASE 3

A married woman, aged 65, was admitted on December 14, 1954, for a discharge from her right ear of one month's duration. One month ago, she had severe pain in her right ear, which lasted for several days. For this, she was given penicillin by mouth for five days. On the third or fourth day of her illness, she had spontaneous discharge from the right ear which had persisted without treatment ever since. She had no headache, vertigo, nausea, or vomiting. An x-ray showed destruction of the right mastoid cells. Cultures from the right ear showed B. pyocyaneus. There was a 40-50 decibel conductive loss in the right ear with presbycusis element by audiogram. A complete blood count, urinalysis, and chest x-ray were normal. On December 15, 1954, a right simple mastoidectomy was performed. The cell partitions were found to be completely destroyed and the lateral sinus was uncovered by disease. The postoperative course was uneventful, and the patient was discharged relieved eight days later.

Comment: This case had been given inadequate doses of antibiotics; only five days of oral penicillin which was stopped in spite of continuance of discharge. Until examination at the hospital no bacteriology had been done. Then a B. pyocyaneus was recovered from the ear culture. In this case there was complete destruction of the mastoid and exposure of the lateral sinus by disease.

CASE 4

A girl, agd 6, was admitted on December 2, 1954, for discharge from the right ear of three months' duration. At the age of two years she had an attack of otitis media. The treatment at this time included a tonsillectomy and adenoidectomy. The patient remained well and free of ear disease until September, 1954, when she had a spontaneous discharge from her right ear, which persisted in spite of "adequate therapy." Local examination of the right ear showed purulent discharge without odor and of a yellowish-white color. The tympanic membrane was markedly thickened and injected. No landmarks could be made out. An anterior marginal perforation was present. The left ear was normal. There was a 30-40 decibel conductive loss of hearing in the right ear. Weber was lateralized to the right. Culture from the right ear showed B. pyocyaneus. An x-ray of mastoids showed the left mastoid normal and the right mastoid underdeveloped. The nasopharynx was negative. The patient was placed on high doses of penicillin and streptomycin and vitamins. Profuse discharge persisted and on December 4, 1954, a right simple mastoidectomy was performed. Postoperatively the discharge gradually subsided with instillation of polymixin powder into the mastoid antrum daily. By December 18 the middle ear was resolved and the mastoid incision was healing in. The patient was discharged on December 20 well healed.

Comment: This patient had had an attack of otitis media four years earlier. Tonsillectomy and adenoidectomy had been done, and since then there was no apparent trouble until the present illness. This time she was given "adequate chemotherapy" without success. No bacteriological determinations were done until her admission to the hospital when bacillus pyocyaneus was cultured from the infected ear. This case was progressing to chronicity with a threatened loss of hearing function.

CASE 5

A boy, aged 9, was admitted on December 6, 1954, with pain in his right ear. This was the first admission for this child, who had had a right earache which "subsided" without treatment approximately one month before admission. Four or five days before this he had pain in the right ear again. He was then admitted for myringotomy and chemotherapy. On local examination the right tympanic membrane was thickened, red, and bulging. There was a lymph node just under the tip of the mastoid. There was slight tenderness elicited on palpation over the mastoid process. The left ear was normal. The urinalysis was normal. Hematology showed slight normocytic anemia. The chest x-ray was negative. An x-ray of mastoids showed the left mastoid normal; there was destruction over the lateral sinus in the right mastoid. A culture from the right ear showed coagulase negative hemolytic staphylococcus aureus. On the day of admission a myringotomy was performed on the right ear, and the pus was sent to the laboratory for bacteriological examination. On December 9, 1954, a right simple mastoidectomy was performed. There was a break through the cortex of the mastoid and the lateral sinus was uncovered by disease. Eleven days later the patient was discharged to his home with his wound healed and the middle ear dry.

Comment: This patient was first seen by an otologist one month after his earache had first appeared. Because this infection had apparently "subsided" without treatment, no antibiotic had been given or myringotomy done early. But by the time of admission the disease had progressed to a complete destruction of the mastoid with exposure of the lateral sinus by disease. This is an example of the complacent attitude that has been apparent lately; that mastoiditis is no longer a disease to be reckoned with and that subsidence of pain and temperature is all that is necessary to serve as a guide.

CASE 6

A boy, aged 15 months, was admitted on November 26, 1954, for pain and discharge of his right ear. This child had been admitted to another hospital five weeks before with a diagnosis of aseptic meningitis. During this admission he was noted to have an infected eardrum and was treated with gantrisin and crysticillin for six and four days respectively and was discharged. He was seen again in the clinic on November 8, 1954, at which time the right eardrum was noted to be very red, but not bulging. He was treated with aureomycin. A follow-up visit on November 10, 1954, revealed a "subsiding otitis media" and the aureomycin was continued. On admission to our clinic November 26, 1954, still on aureomycin, the mother gave a story of a convulsion on the previous day and also stated that the child had fallen to the right side several times that day. The patient was

admitted to the pediatric service of the Massachusetts General Hospital and the
otological service was consulted immediately. Examination revealed a tympanic
mmberane without landmarks, thick, dull, but with fair mobility and not bulging.
An x-ray of the mastoids revealed definite destruction in the right antral region.
A myringotomy was performed on the right side and much pus was obtained,
which was cultured. This was reported to be coagulase positive hemolytic staphylo-
coccus aureus. The patient was operated upon on the following day for a right
simple mastoidectomy. The intracellular partitions appeared to be broken down
and filled with granulations and pus. Postoperatively the patient did very well.
The drum had returned to normal by December 6. The postauricular incision was
closed by December 13.

Comment: In this case there was an apparent reluctance to
consider the infection from the surgical point of view at the onset.
Chemotherapy or antibiotics were given without much regard for the
bacteria and without due consideration that surgical drainage was
likewise necessary.

<center>CONCLUSION</center>

It is the conclusion of the authors that the chief causes for these
patients' coming to operation were first, the attitude that acute otitis
media carries no great implications of danger; that merely arresting
the pain and temperature by antibiotics is all that is needed to obtain
a cure. One may call this the attitude of complacence; second, a
reluctance to consider suppurative otitis meia as a surgical disease. It
is quite obvious that in any case of retained pus, drainage is necessary,
for as Fleming has pointed out the drug must come in contact with
the bacteria directly. The presence of undrained purulent secretion
frequently will prevent just such an occurrence. Also in the authors'
opinion, the best bacteriological determination can be obtained from
a culture taken directly from the middle ear secretions at the time
of myringotomy. They would like to stress also the importance of
taking a culture prior to the administration of penicillin or other
antibiotic drugs. If one wishes to give any drug during the interval
between the taking of the culture and its reporting, sulfadiazine may
be given which does not have the effect of inhibiting the growth in
vitro. Finally, it is pointed out that at the present time many or-
ganisms, principally strains of staphylococci, have developed specific
resistance to one or more of the antibiotics. Furthermore, organisms
such as B. pyocyaneus and H. influenzae have appeared in increasing
numbers. It would seem obvious that bacteriological studies are es-
sential and that on certain occasions sensitivity tests must be done to
select the proper antibiotic. If these principles are not considered
we may be in for an increase in the number of mastoidectomies, a
reversal of the trend noted up to the present time. One wonders why
there seems to be such opposition to a simple myringotomy, such

fear of "traumatizing" the drum with disregard of the real danger from the presence of purulent exudate in the middle ear with its implication of loss of hearing function, or the possible extension of disease to other regions of the ear.

REFERENCES

1. Tobey, Harold G.: The Sulfonamide Drugs in the Treatment of Acute Otitis Media, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 51:945-954 (Dec.) 1942.

2. House, Howard P.: Otitis Media: A Comparative Study of Results Obtained in Therapy Before and After the Introduction of Sulfonamide Compounds, Arch. Otolaryng. 43:371-378 (Apr.) 1946.

3. Page, J. R.: Bacteriology of Acute Infections of the Middle Ear and Mastoid, Arch. Otolaryng. 20:447-451 (Oct.) 1934.

4. Richardson, John R.: Observations of Acute Otitis Media, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 51:804-816 (Sept.) 1942.

5. Butler, Harry: Low Incidence of Mastoiditis in Series of Cases Treated Without Sulfonamids, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 57: 992-998 (Dec.) 1948.

6. Evans, M. G.: The Treatment of Acute Suppurative Otitis Media. The Relative Merits of Chemotherapy and Myringotomy in Avoiding Surgical Mastoiditis, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 60:638 (Sept.) 1951.

7. Zonderman, Bernard: Modern Concepts in the Treatment of Otitis Media, New Eng. Jour. of Med. 249:643-645 (Oct. 15) 1953.

8. Davison, F. W.: Antibiotics and Otolaryngologic Infections, Bull. Geisinger Memorial Hosp. and Foss Clinic 6:4 (Nov.) 1954.

9. Fleming, A.: Penicillin, Its Practical Application, The Blakiston Co., Philadelphia, 1946.

XXII

MANAGEMENT OF ALLERGIC SINUSITIS

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Successful management of allergic sinusitis is contingent upon close co-operation between the allergist and the rhinologist.

Allergists, in treating respiratory allergy, are often prone to minimize the import nce of rhinologic examination and therapy, and rely on elimination, injection, and antihistamine therapy despite the presence of polyps and symptoms referable to the sinuses.

Similarly, otolaryngologists too often fail to realize the importance of the role played by allergy in the inception and maintenance of attacks of sinusitis. Because of this many patients are deprived of proper allergy survey and along with this group are those who fail to receive an allergy study because facilities for such a study are not always readily available.

This handicap is overcome by otolaryngologists who are equipped for and do supervise their own allergy studies, and this has proven to be a highly satisfactory method wherever it has been put into practice. If there are disadvantages associated with this sort of practice they are more than outweighed by the advantages gained. In this manner the patient is kept under constant surveillance by one qualified to observe and study the changes in the nasal mucosa and to render such local treatment as is indicated from time to time.

PATHOLOGY

Nasal and sinus allergy is in reality a catarrhal inflammation characterized by tissue edema with eosinophilic infiltration and a muco-serous discharge of glandular and goblet cell origin. The edema may be transitory and disappear with the removal of the irritant. If the irritant persists and the edema becomes pronounced the stroma becomes overloaded and the tissue prolapses into the nasal or sinus cavity in the formation of a polyp.

Polyps are prone to develop in the areas of more delicate tissue construction. The loosely bound strands of fibrous tissue character-

Fig. 1.—The specimen illustrated shows a polyp which has arisen from the upper attachment of the uncinate process. This is a common site of origin.
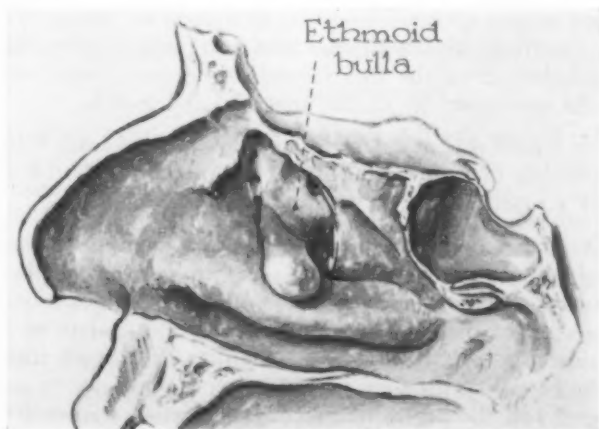
Fig. 2.—The specimen illustrated shows a polyp arising from the anterior face of the ethmoidal bulla. This area and that shown in Figure 1, the uncinate process, are the two most common sites of origin of nasal polyps.

istic of the mucosa of the middle meatus is conducive to the collection
of edema and the development of polyps. Polyps commonly arise
from the margins of sinus ostia, the crest of the ucinate process (Fig.
1) and the anterior face of the bulla ethmoidalis (Fig. 2). Contrary
to a generally accepted belief these growths do not originate from
within small cells. The thin membrane lining the ethmoid sinuses
may become edematous to the point of completely obliterating the
lumen of the cavity and polyps may arise from the margins of the
ostia and drainage grooves of the cells but the prolapse is always into
the adjacent meatus.

Edema in chronic nasal and sinus allergy is usually asso-
ciated with a pale allergic exudate in the sinus cavity. This collects
in the nose and nasopharynx and is a source of considerable annoy-
ance.

Acute flare-ups of a purulent nature are to be expected in these
cases due to impairment of sinus drainage and this is especially so in
the presence of blocking polyps.

SYMPTOMS AND DIAGNOSIS

The usual symptoms of nasal allergy are those of nasal blockage
with clear watery discharge, sneezing attacks, itching of the eyes
and nose, pale postnasal discharge, and a feeling of congestion in the
upper straits of the nose. This may be described by the patient as a
pain extending over the frontal area.

Any or all of these symptoms may be present with the addition
of others such as occipital headache, hoarseness or huskiness, chronic
cough, catarrhal otitis with flightiness or vertigo, pains about the
face extending down the arm (Sluder's syndrome), edematous areas
about the face especially of the upper lip and eyelids.

The typical patient complains of frequent head colds with minor
symptoms but these are inclined to become more pronouced with the
onset of a superimposed suppurative sinusitis.

The diagnosis of nasal allergy is made on the history, family and
personal, the presenting symptoms, the results of nasal smears and
skin testing. Rhinologic examination, anterior and posterior, reveals
the character of the nasal tissues, the presence of polyps or pus and
the architecture of the intranasal structures. Roentgen studies dis-
close the nature of the sinus involvement, the presence of polyps or
fluid level and the degree of mucosal thickening. Sinus lavage by
cannula or displacement reveals the character of the sinus exudate.
A pale exudate containing a preponderance of eosinophiles is indica-
tive of a nonpurulent allergic sinusitis, a yellowish mucopurulent
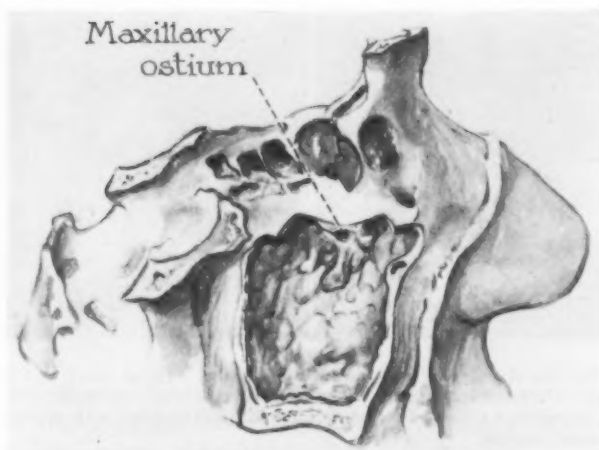
Fig.3.—In the specimen illustrated the lining mucosa of the maxillary sinus shows allergic polypoid edema with small polyps in the neighborhood or the maxillary ostium.

discharge contains many neutrophiles and is characteristic of an acute purulent attack while an exudate with a greyish or greenish color with a preponderance of lymphocytes and plasma cells denotes chronicity. Exudate with a foul odor is often found in a completely blocked sinus or it may be associated with an infection of dental origin.

ANATOMIC CHARACTERISTICS

Maxillary sinus. The maxillary ostium is as a rule of ample size and the infundibulum is sufficiently wide to permit an unhampered egress from the sinus of large clumps of heavy exudate. In cases with a normally functioning ostium, the ciliary mechanism carries the secretions up the sinus wall through the opening and out into the middle meatus as rapidly as they accumulate. Drainage barriers at any point along the line, cause a stasis of secretions with a tendency toward persistence of infection in the cavity. In some of the long-standing cases the fault is in the ostium itself. This is revealed at the time of irrigation, as the restricted ostium prevents a free return flow of the irrigating solution (Fig. 3).

This, in certain instances, is due to the presence of a polyp in the ostium or in the sinus which presses valvelike against the ostium, but in most cases the opening is constricted by swelling of the osteal mu-

Fig. 4.—Roentgenograms of sinuses showing typical nasal and sinus allergy. A. Scroll-like appearance of edematous inferior turbinates. B. Allergic appearance of antral mucosa with nasal cavities filled with polyps and edematous mucosa.

cosa which is involved in the same edematous process that affects the entire membranous lining of the cavity. In many of these cases, the mucosa lining the infundibulum is also involved; in some, to the extent of a complete sealing off of this channel. In other cases the middle meatus may be filled with polyps or blocked by an impinging middle turbinate.

Frontal sinus. The frontal ostium located in the most dependent portion of the sinus floor and opening as it does in a majority of cases directly into the middle meatus provides ideal drainage facilities. These may be hampered by an impinging middle turbinate or encroaching frontal cells either within the sinus or adjacent to the opening in the middle meatus.

Ethmoid sinus. Ethmoid cells commonly drain into grooves. These form fairly parallel lines along the face of the upper lateral nasal wall. They are the infundibulum and the bullar ostium in the middle meatus, the posterior groove in the superior meatus and when present (67 per cent) the postreme groove in the supreme meatus.

Groups of cells having a common drainage space should be similarly involved in an infective process. It is for this reason that the maxillary sinus, the infundibular cells and occasionally the frontal sinus are simultaneously infected. For the same reason buller cells which have a separate drainage ostium often escape an infection involving all the other anterior cavities.

The bullar cells have the poorest drainage of all the ethmoid groups. Unlike the others which have dependent drainage it may in these cells be impaired, but seldom to the point of complete occlusion. This may occur, however, with the formation of polyps which arise from the margins of the sinus openings and from the overhanging edges of the superior and supreme turbinates.

Sphenoid sinus. Infections in the sphenoid sinus usually exist unrecognized and unsuspected for many months or years. When they finally come up for investigation, the ostium is more than likely narrowed by chronic edema. This condition is suspected if on irrigation the return flow is retarded. In most of these cases thickened mucosa also is present adjacent to the ostium on the anterior face of the sinus.

TREATMENT

A fundamental requirement in the proper management of allergic sinusitis is control of the allergy. Without this, improvement is possible but a return of the condition is to be expected.

In an allergic state the sinus mucosa is edematous in varying degrees and the lining of the sinus ostium is similarly involved. This results in a partial to complete closure of the opening, inhibiting its normal function as a drainage outlet. With the allergy under control, edema diminishes, the ostium opens up and the sinus secretions are waved into the nose by the cilia.

Elimination of offenders. Successful management of respiratory allergy calls for elimination as completely as possible from the patient's environment, the allergens to which he is sensitive. This may require the removal of pets, a dust-proof cover for the pillows and mattresses, elimination of cosmetics containing orris root, replacement of woolen bed covers with those of other material, the use of oil in the dust cloths and mops, and in certain cases, removal of woolen floor coverings. Foods known to be offenders must naturally be avoided as well as freshly painted rooms and irritating fumes such as those given off by DDT insect spray. Tobacco smoke must be entirely avoided by some individuals who cannot remain in a room containing even a small amount of smoke.

Some patients remain problems despite the fact that they have received all possible attention while on the other hand an apparent problem is often solved by the chance discovery of contact with a previously overlooked offender.

Some individuals find it necessary to change occupations for the dust associated with their work is the disturbing factor while others do better after moving to a different climate.
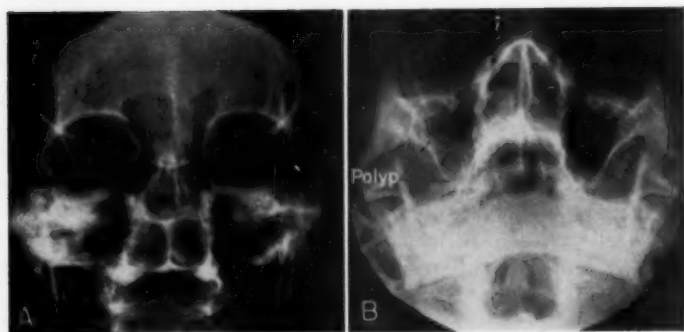
Fig. 5.—Roentgenograms of sinuses showing: *A.* Edematous membranes lining nasal cavities. *B.* Same case as in *A*, showing shadow of large antral polyp.

House dust therapy. Injections of extract made from a composite collection of dust from various houses is widely and sucessfully used in producing hyposensitization in individuals allergic to house dust. This especially has been the case since the introduction of the low-dosage technique suggested by Hansel. The usual form of therapy is an injection once or twice a week, beginning with a dilution of 1:1,000,000 or even weaker and gradually increasing the strength until a relief of symptoms is experienced. The immediate goal is to reach the optimum dosage which will produce an alleviation of symptoms and maintaining it at that point while the period between the injections is gradually widened.

Sinusitis. During this whole procedure treatment of the sinuses is carried out in similar manner to that rendered in the nonallergic case.

Most cases are comparatively simple and a satisfactory diagnosis is reached and proper treatment introduced at the time of the initial visit. This is not always possible and many visits and diagnostic measures may be required before the exact nature and extent of sinus involvement is determined. In some instances the exact diagnosis may never be reached; as the allergy becomes effective the symptoms referable to the sinuses are alleviated and further diagnosis and therapeutic procedures, for the time being at least, seem unnecessary. Some cases, however, persist and require a painstaking study and careful planning with a therapeutic regime covering a considerable period of time.

CLINICAL CLASSIFICATION

A wide variety of cases of allergic sinusitis is commonly observed but these roughly may be classified into five major clinical groups.

1.   Acute suppurative sinusitis on an allergic basis.

2.   Subacute sinusitis maintained by structural drainage barriers and allergic edema.

3.   Acute and subacute sinusitis with drainage blocked by polyps.

4.   Chronic sinusitis with edematous constriction of sinus outlet.

5.   Chronic sinusitis with or without polyposis which has been subjected to one or more radical surgical procedures.

1.   In the first group are those individuals who suffer from frequent head colds which usually result in acute attacks of sinusitis. In these cases the diagnosis of allergy has not been made although no doubt suspected as being a contributing factor at various times. The cause of the sinus involvement is acute edema which prevents a free flow of secretions from the cavity. These cases respond well to irrigation therapy as the edema subsides or the attack may clear up within two or three weeks without treatment, and if hyposensitization is attained the frequency and persistency of the attacks will be minimized.

2.   In the second group the cases resemble those in the first group except that the sinus attacks in these cases persist even after the subsidence of the edema. Most of the patients in this group have had their sinuses treated at various times and received only temporary relief. The condition always returns and they have come to regard themselves as "hopeless sinus sufferers." Successful management in cases of this type include correction of drainage barriers as well as allergy therapy.

3.   In the third group most of the individuals are cognisant of the fact that allergy is the basis for their recurrent polyps and sinusitis although many have failed to receive proper study and treatment. With polyposis there usually is an associated ethmomaxillary sinusitis. With the nose free of polyps these cases respond well to displacement therapy with antral irrigation when indicated. The polyps and sinusitis will continue to recur unless the allergy is kept under control.

4.   In long-standing cases of allergic sinusitis there is a tendency toward occlusion of the ostia by chronic edema. This especially applies to the sphenoid and maxillary sinuses, both with drainage high on the sinus wall near the roof of the sinus. Characteristic of both of these sinuses, in cases of this type, is the extension of the edematous
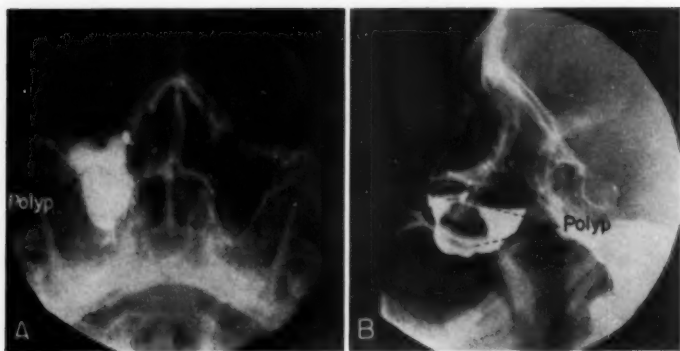
Fig. 6.—Roentgenograms (same case as Figure 5) after instillation of opaque medium in the maxillary sinus. Large polyp causes filling defect.

process to the structures in the nose adjacent to the ostium. In these cases little can be accomplished toward restoration of the drainage passages of the sinuses. In certain cases of sphenoid involvement collections of thickened mucosa adjacent to the ostium may be removed with biting forceps and this followed by a few irrigations may temporarily rid the sinus of infection but there is no assurance of permanent relief and a window may be indicated. This is placed on the anterior face of the sinus wall below the ostium either by direct approach through the olfactory fissure or through the transeptal approach and this supplies, in most instances, an adequate permanent drainage outlet.

The maxillary sinus is treated in similar fashion. If early relief is not attained by unblocking the middle meatus and irrigation therapy a window is placed in the inferior meatal wall. The success achieved by this measure when combined with control of allergy, is widely acknowledged and more and more it is replacing the canine fossa approach in the management of chronic maxillary sinusitis.

5. The cases which present the real problem in sinusitis are those which have been subjected to radical surgical procedures at one time or another. Most of these procedures on the sinuses are instigated because of long-standing infections with thickened sinus mucosa which have failed to respond to a few irrigations or because of polyps or polypoid tissue in the middle meatus.

The procedures are aimed at removal of thickened membranes and growths through a Caldwell-Luc operation and intranasal ethmoid exenteration.

Following these operations there usually is a period of freedom from polyps and annoying symptoms. This in most instances is not permanent for the factors which caused them have not been eliminated and their eventual return is to be predicted. The antral cavity again becomes lined with a membrane subject to edema and reinfection and nothing has been accomplished by the Caldwell-Luc unless in the process a large antral polyp has been removed or unless the technique has included a serviceable nasoantral window. In the middle meatus following a removal of cells, the area sooner or later fills up with edematous tissue. Regardless of the approach or surgical skill employed, scar tissue forms to block drainage of remaining cells and to hide away pockets of infection, and to that may be added the new rough bony surfaces now available for the development of polyps.

Comment. In many of the cases in this final group very little attention has been paid to allergy as a possible contributing factor to the cause of their condition, yet most of the patients who come under this heading are allergic to inhalants of one form or another. In some this has been determined but in very few has a concentrated effort been made toward a control of the allergy as a preoperative measure.

MANAGEMENT

With a return of edematous tissue and infection little is to be gained by repeated surgical procedures. Much may be accomplished, however, by measures to aid in lessening the symptoms and the correction of local and systemic contributing factors. A plan of study should be outlined and this should include aside from the allergy survey, a consideration of possible gastrointestinal, endocrine and nutritional disturbances and infections elsewhere. Also in order may be a study of the patient's habits, indulgences and psychic make-up. Many patients fail to make much progress and may even retrogress because of undue emotional strain and physical fatigue. In such cases the adaptation syndrome and alarm reaction of Selye[1] may be applied. Selye has demonstrated that there are normal physiological and biochemical reactions of the body to shock and stress. The powerful hormones of the adrenal cortex become depleted or are liberated to excess and this results in profound changes in various body structures. Much progress has been made recently in our understanding of the mechanisms of these hormones, their influence on inhibiting allergic edema and inflammation, fibrosis and granulation tissue. Corticotropin, cortisone and hydrocortisone have a wide range of application in the

field of otolaryngology and it could well be that as our knowledge concerning these hormones increases they may be applied successfully in the management of allergic sinusitis in its various forms.

The patients in group five when examined rhinologically usually reveal wide-open nasal spaces with polyps or edematous membranes in the middle meatus and streaks of exudate in the folds of tissue, collecting in the nasopharynx. In most cases the polyps may be removed to aid in restoring breathing and drainage space. In certain cases, those who have had turbinectomies and other mutilating operations on the nose, polyps may best be left intact for they are merely serving in a compensatory space-filling capacity.

In most of the cases the displacement measure offers the best solution to the problem of retained nasal secretions and many patients feel fairly comfortable with an occasional such treatment while allergy control along with the correction of all other possible disorders is being achieved.

SUMMARY

Successful management of allergic sinusitis is contingent upon control of the allergy.

Whether or not the rhinologist supervises the allergic management of his cases, he should see his patients periodically for examination and treatment.

Edema is the outstanding characteristic of allergy and its effects in rhinology are many-fold. Allergic edema and polyps block the nose and diminish the size of the sinus drainage outlets to a minimum thereby aiding in the inception and maintenance of attacks of sinusitis.

Polyps arise from rough bony surfaces and edges as well as the margins of sinus ostia. They do not come from within ethmoid cells and these cells should be left intact in the operation of polypectomy.

Cases of allergic sinusitis may be classified roughly into five groups. They include the cases of acute suppurative sinusitis on an allergic basis, those with structural defects and polyps blocking sinus drainage, those with chronic edema constricting the ostia and the cases of chronic sinusitis with or without polyposis which have been subjected to one or more radical surgical procedures.

Treatment of the various types of allergic sinusitis is carried out in similar manner to that rendered in the nonallergic case with the goal of the rhinologist ever being the establishement of adequate sinus drainage passage ways.

Radical surgical procedures are seldom indicated in allergic sinus-itis. Allergic membranes are replaced by membranes also allergic and the patient's symptoms although relieved temporarily may return along with others and perhaps more pronounced than they were previously.

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REFERENCES

1. Selye, H.: The Physiology and Pathology of Exposure to Stress: A Treatise Based on the Concepts of the General Adaptation Syndrome and Diseases of Adaptation, Montreal, Canada, Act, Inc., 1950.

2. Rawlins, A. G.: The Mesenchyme of the Nose and Sinuses, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 62:307-315 (June) 1953.

3. Rawlins, A. G.: Corticotropin and Cortisone in Otolaryngology, J. A. M. A. 157:500-502 (Feb.) 1955.

XXIII

CHOLESTEATOMA OF THE TEMPORAL BONE

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Cholesteatoma of the middle ear cavity and mastoid have as a rule presented few problems to the otologist. A complete and thorough radical mastoid operation has been sufficient to control these cases. Serious problems arise, however, when cholesteatoma involves other regions of the temporal bone than the mastoid and the middle ear cavity. Two such cases are presented which illustrate the difficulties involved in attempting to cure these cases. One is that of a direct invasion of the middle cranial cavity; the second case is that of an involvement of the petrous apex and the internal auditory canal.

REPORT OF CASES

CASE 1.—A forty-two year old male was seen in September, 1946, with swelling and pain of the left external auditory canal for two weeks following swimming.

At the age of nine (1913) he had had a left radical mastoid operation, with a skin graft. Since then, he had been well and had achieved outstanding success in his work. For the past five years he has had occasional attacks of pain and slight swelling in the left temporal region accompanied by queer sensations which he could not describe. For the past year, he has found it difficult to carry on his work on account of inability to concentrate, with episodes of depression and forgetfulness. These attacks had increased in intensity so that he stopped his work and was being treated by a psychiatrist for this complaint. At no time in the past year had any episode occurred that would center attention on his ear. The episodes of depression were blamed on the complex and disturbing business problems of his profession.

Examination: The right ear was normal; the left ear showed a retro-auricular scar of the radical mastoid operation, a slight foul discharge from the cavity, slight swelling of the temporal muscle region and no fluctuation or pain. The auricle was normal. On cleaning the radical cavity a cholesteatomatous mass was removed and the tegmen was found to have been absorbed and replaced by
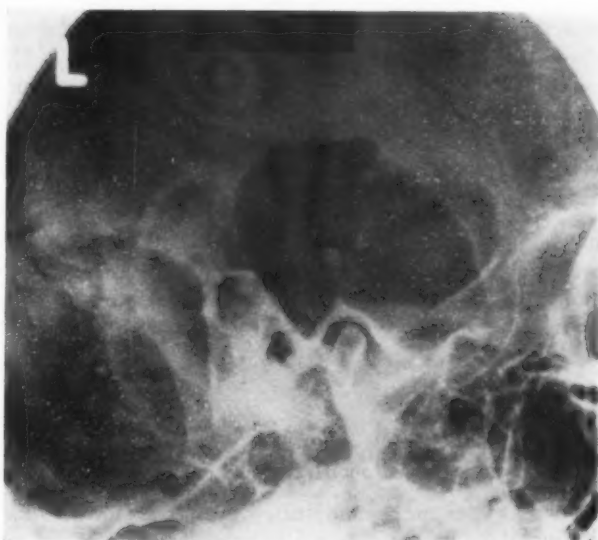
Fig. 1.—Roentgenogram, Case 1. There has been a radical mastoid operation on the left side, and there is a large area of destruction about the squamous part of the temporal bone, the posterior part of the parietal bone and part of the greater wing of the sphenoid. The outline is smooth, suggesting a possibility of an epidermoid growth.

cholesteatoma. X-rays showed a marked deficiency of the squamous portion of the temporal bone (Fig. 1). The middle ear was clean. There was no facial paralysis. The hearing test showed the right ear to be normal. The left ear had a loss of 35 db for 128, 256, 512, and 1024; a loss of 60 db for 2048 and 90 db for 4096. The patient stated that he used his left ear in telephoning without any difficulty. The eye-grounds and fields were normal. All neurological reflexes were normal.

One week later, under general anesthesia, the left mastoid was opened through the postaural incision, and a mass of cholesteatoma was seen lying above the radical cavity and extending forward. A temporal muscle decompression was then done, the incision running from the external angle of the orbit to join the postaural incision. The temporal muscle and the auricle were then turned down and forward exposing the great wing of the sphenoid, the posterior portion of the frontal and the anterior portion of the parietal bone.

A mass of cholesteatoma about 5 cm in diameter was exposed protruding above the edges of the absorbed bone. This was removed en masse and was found to extend into the middle cranial fossa, having pushed the brain aside for about 5 cm in depth (Fig. 2). The dura was intact, but so thin and translucent that the gyri and sulci of the temporal lobe of the brain could be seen easily. On removal of the mass the brain immediately filled the cavity and bulged slightly over the edge of the bony defect. The dura was then stripped of all the epithelium left on it without tearing it (Fig. 3).

The cholesteatoma was found to have extended under the zygomatic process, being attached to the temporal muscle and exposing the capsule of the left jaw joint. This was cleaned out and a layer of temporal muscle, with cholesteatoma attached to it, was removed.

The radical mastoid and middle ear cavities required no operative procedure and were let alone.

The temporal muscle decompression was then closed and the postaural incision was left open and packed with iodoform gauze.

Two weeks later the postauricular wound was closed and a plastic operation was done on the external auditory canal to give a wide approach to the operated area.

The patient had a prolonged and slow convalescence. The discharge gradually stopped and the small areas of cholesteatoma seen on the tegmental portion of the dura were cleaned off as they appeared. The mental symptoms gradually cleared up, and the patient began to take an active interest in his profession. About one year after the operation, in 1948, he again resumed active work and has been very successful.

From 1948 on, the patient was seen on an average of once every four months. The radical cavity was cleaned and an occasional thin layer of cholesteatoma would be peeled off the dura which was now normal in appearance.

November, 1954. Examination of the patient showed a slight separation of the dura from the overlying tissue, and with mirrors and the nasopharyngoscope, a thin layer of cholesteatoma was seen in the parietal region. For this reason and because of a history of occasional slight headaches in the preceding two months re-exploration of the temporal muscle region was advised.

December, 1954: Under general anesthesia, a temporal muscle decompression was done through the original incision. The mastoid was not exposed postaurally. On turning the flap down a layer of
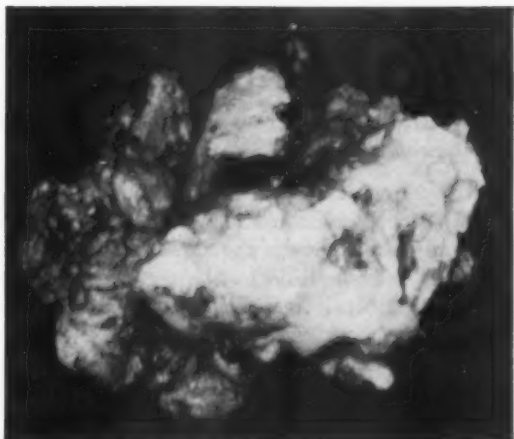
Fig. 2.—The mass of cholesteatoma removed, averaging 5 cm in diameter.

Fig. 3, Case 1.—The appearance of the brain as it bulged through the decompression wound when the cholesteatoma had been removed. Note the dipping in of the dura in the region of the sulci.

cholesteatoma about 2 mm thick was found lying in the upper part of the exposed dura. This was removed and the dura scraped with curettes; the temporal muscle again was stripped and cleaned of cholesteatoma and the wound was closed. Ten days after the operation the patient was discharged from the hospital.

The hearing has remained the same throughout the past eight years, and the patient has returned to work again.

The problem presented by this case is the impossibility not only of removing all the cholesteatoma found but in externalizing the cavity, and the only alternative is the constant watching of the patient, with possible re-exploration of the region if symptoms began to reappear. The patient is reconciled to the prospect of having another exploration in about eight to ten years.

CASE 2.—A forty year old male was admitted to the hospital on January 20, 1944, with a diagnosis of partial right facial paralysis and a question of brain tumor. He complained of headaches and weakness of the right side of the face.

Eight months previously the patient had fallen from a car to the ground and struck his right elbow, shoulder and the back of his head. This dazed him for a few minutes. He was not unconscious, therefore he got up and started work again. He had no headaches, nausea or dizziness afterwards.

Three weeks later he noticed a twitching of the right side of his face. The twitching gradually stopped, but four months before admission he had a definite weakness of the right facial muscles. At the same time he began to have headaches. The headaches started from the right frontal region and radiated to the right side and back of the head. They lasted several days but were relieved by aspirin and eating. There was still no nausea, vomiting, dizziness, or tinnitus; however, he did notice a loss of hearing in the right ear.

Examination showed a slight tenderness of the right temporal region. No glands of the neck were palpable. The fundi were normal. There were no visual defects, and corneal reflexes were present. There was no nystagmus. There was a right facial weakness. The patient's mouth opened without distortion. His tongue protruded in a straight line. The knee and ankle jerk were within normal limits.

Ears. Examination of the left ear showed scarring of the epitympanic space. The drum was thickened and retracted. There was no discharge. Examination of the right ear showed an old chronic otitis with a large epitympanic perforation. There was no discharge and no evidence of cholesteatoma in the region of the perforation.

A hearing test showed in the left ear a typical middle ear deafness with a loss of 35 db up to 3000. The right ear, with masking of the left ear, showed a loss of 80 db from 512 to 5000. He did not hear 128 or 256. Weber was to the left.

Barany Turning Test: On turning the patient to the right or left a response of 20 seconds was obtained in both directions. A diagnosis was made of a dead right labyrinth, secondary to trauma or cholesteatoma.

X-rays show the bones of calvarium normal; the sella turcica was normal; the pineal gland was calcified and not displaced. The right petrous bone showed a large area of diminished density superiorly and posteriorly, extending almost to the petrous tip. The wall of the petrous tip was thinned to almost a line over it, the margin was smooth but showed a slightly increased density. The internal meatus was not well visualized but did not appear to be enlarged.

The mastoids were sclerotic. The findings were those of an area of destruction in the central portion of the right petrous bone. There was almost certainly either a cholesteatoma, an epidermoid growth, or a neuroma of the acoustic nerve located laterally to the usual position and entirely in the petrous bone. The appearance of the mastoid indicated an old chronic infection without evidence of any recent destruction (Figs. 4 and 5).

The ventriculogram gave no evidence of any brain tumor. There was a normal filling of the lateral and the third ventricles without evidence of dilation or displacement. There were no filling defects. Ventriculograms were negative.

A radical mastoid operation with exploration of the right petrous pyramid was advised.

February 8, 1944. At the operation it was decided to let the facial nerve alone, as there was not a complete facial paralysis. It was hoped that with relief of pressure on the facial nerve its function might return. With this in view a Neumann operation was done.

The usual postauricular mastoid incision was made. The semicircular canals were exposed and the bony vestibule entered through the horizontal canal. Cholesteatoma was found in the bony vestibule. The oval and round windows were then connected and the cholesteatoma of the vestibule was removed. With removal of the cholesteatoma the internal auditory canal was found open and there was considerable leakage of cerebrospinal fluid. The leakage was controlled with muscle packs from the temporal muscle. The cavity
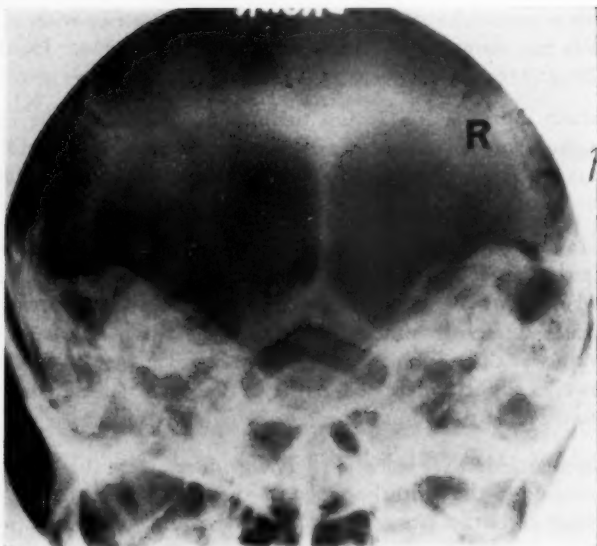
Fig. 4.—Roentgenogram, Case 2. The left mastoid is underdeveloped and sclerosed. The right has been operated upon. There is destruction of the cochlea and the semicircular canal and of the petrous pyramids beyond this area up to the internal auditory meatus.
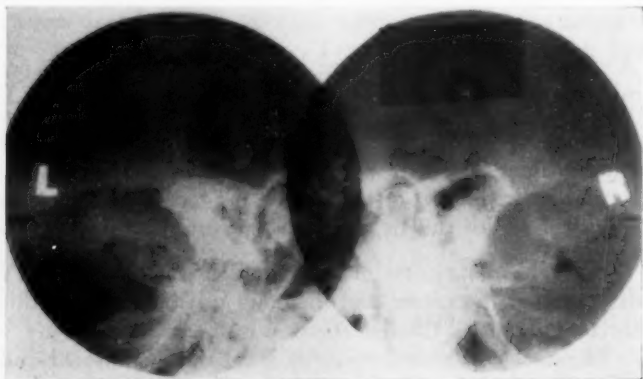
Fig. 5.—Roentgenograms, Case 2. There is a clean cut cholesteatoma cavity in the petrous pyramid, in the region of the internal auditory meatus. There has been destruction of the cochlea, on the right side. The left mastoid is completely sclerosed.

was cleaned of cholesteatoma as much as possible, and the wound was packed with iodoform gauze.

The leakage of cerebrospinal fluid gradually stopped, so that on the fifth day after the operation the wound remained dry.

Ten days after the operation all packs were removed and the postauricular wound was closed. The facial paralysis was now complete.

From 1945 until 1954, there was no evidence of any recurrence. The facial paralysis was still complete but the patient otherwise did very well. In December 1954, he complained of slight heaviness of the right side of the head. Examination of the cavity showed a small lead from the exenterated vestibule extending toward the apex of the temporal bone. On probing the lead a small amount of cholesteatoma was removed. The facial ridge being present prevented a direct exploration of the cavity found. It was advised that the cavity be re-explored and revised so that the petrous apex could be watched under direct vision.

January 17, 1955. Eleven years after the first operation the right temporal bone was re-explored. The usual endaural incisions were made and the radical mastoid cavity was widely exposed. The facial ridge and facial nerve were removed down to the stylomastoid foramen. The bony cochlea was exenterated, the jugular bulb and the carotid artery were exposed. The internal auditory canal was filled with dense fibrous tissue, and there was no evidence of any cerebrospinal fluid leak. Cholesteatoma was found extending from the vestibule to the apex of the temporal bone and was removed. There was a thin layer of bone separating the cholesteatoma cavity from the Gasserian ganglion, and from the dura of the posterior cranial cavity. The cholesteatomatous lining was completely removed with curettes. The cavity was then packed with iodoform gauze and the patient returned to bed in good condition.

Ten days following the operation he left the hospital, reporting back once a week for repacking of the cavity.

In this case the chief error lay in trying to save the facial nerve; if the facial ridge had been taken down at the first operation, there would have been less difficulty in taking care of the cavity and watching for recurrence of the cholesteatoma. Opening the internal auditory canal and packing it to control loss of cerebrospinal fluid, was also a factor in preventing a complete and thorough removal of the cholesteatoma. In all probability some cholesteatomatous matter was left in the region of the internal auditory canal, which was able to

regenerate under the granulations present.   This regeneration was
extremely slow for there was an interval of ten years before symptoms
began to recur.

These two cases are reported to call attention to the fact that
cholesteatomas outside of the mastoid cavity in the temporal bone
are difficult to eradicate and that one must constantly keep in mind
possible recurrence, and be ready to re-explore when symptoms re-
cur, even though the cavity when examined may appear not to be
diseased.

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XXIV

INDICATIONS FOR NECK DISSECTION IN MALIGNANCY OF THE THYROID

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With a desire to show in a small way my deep appreciation of a long friendship over the years, and my admiration and esteem for Doctor Mosher, I would like to discuss the necessity of neck dissection in malignancies of the thyroid, with a report of a few cases taken at random to show the various types of malignancies best suited for this procedure. It had been suggested to me that my paper be on a subject with which I had become identified over the years. However, in the past I have written so much about carcinoma of the larynx, pharynx and oropharynx, with the need of neck dissection in the more extensive types, that to write about them again would, I fear, be repetitious.

Almost thirty years ago I had the opportunity of continuing in Dr. Mosher's laboratory at the Massachusetts Eye and Ear Infirmary, dissections of the neck as I had learned them from Sir Wilfred Trotter of London.[1] I[2] am indeed grateful to have had the good fortune to work under the guidance of these two renowned gentlemen, and I am deeply indebted to them for whatever success I have attained through their help.

We are all fairly in accord with the progress made as to the advisability of neck dissection in carcinoma of the sinuses, nasopharynx, oropharynx, pharynx, and the extensive lesions of the larynx.[3] The same should be with regard to carcinoma of the thyroid.

In carcinoma of the larynx, the operative procedure has been extended by including the radical neck dissection;[4] the resection of the cervical nodes, fascia, muscle, ligation of the internal jugular vein, on one or both sides, at the time of the original operation, or later, at the first instance of a cervical node. The same procedure should apply in malignancy of the thyroid.

Malignancies from the thyroid may spread by contiguous structure, to the carotid sheath, by the blood stream, or by direct extension to the trachea or esophagus.

As in all malignancies of the head and neck, one should be versed in the lymphatics in this area.

We are all fully aware of the frequency with which patients first consult the doctor for a lump in the neck as their first symptom of trouble in the upper respiratory tract; this also applies to the thyroid. To successfully cure there patients, if the primary lesion is operable, is to know thoroughly the lymphatic drainage of the head and neck. For example: a patient (Case 1) consulted me for a lump in the neck which was movable and nodular, situated under the sterno-mastoid muscle at the level of the bifurcation of the carotid artery. No evidence of any malignancy was seen in the nose, nasopharynx, sinuses, oropharynx, pharynx, or larynx. Aspiration biopsy revealed a papillary cystadenocarcinoma of the thyroid, which proved to be metastatic. At operation, the primary growth was in the thyroid gland on the posteromesial aspect of the gland which had become firmly attached to the trachea.

To summarize, the lymphatic drainage of the thyroid is as follows: The lymphatics[6] of the mesial area of the superior pole and within the upper portion of the isthmus, drain into the prelaryngeal and thyrohyoid nodes, and finally reach the deep cervical chain.

The lateral area of the superior lobes drain the anterolateral and posterolateral surface of the thyroid and pass upwards laterally in front of, and behind the carotid artery, to terminate in the deep cervical chain.

From the inferior portion of the gland the lymphatics drain into the paratracheal and pretracheal nodes to terminate in the deep cervical chain. Direct drainage from the lymphatic vessels into veins, with or without passing through lymph nodes, has been demonstrated. The deep cervical chain receives ultimately all the drainage from the head and neck.

We must remember that the flow of lymph following a resection may have a retrograde movement, involving the nodes in the post-auricular area, posterior triangle of the neck, ending in the retro-pharyngeal nodes.

When we have a patient who has the entire length of the deep cervical chain of lymphatic nodes involved in a fixed, hard, firm mass, extending to a point below the infra-omohyoid muscle, it is needless to say we cannot expect to cure the cancer. By that time the cells have already entered the thoracic duct and gone into the general blood stream; especially when this is accompanied with marked loss of weight, dysphagia, dyspnea, with immediate indications for gastros-

tomyor tracheotomy, the prognosis is hopeless as to cure or even as to appreciable palliative relief.

Complete destruction of all cancer cells is at present the only known method of effecting a permanent cure, whether this be by surgery, irradiation or some biologic change in our make-up.

What constitutes a radical neck dissection? In malignancy of the thyroid, the incision in the neck should be one that will expose both the anterior and the posterior triangles of the neck on the affected side, from the midline of the neck to the anterior border of the trapezius muscle. The dissection includes all the fascia, the prethyroid muscles, including the sternohyoid, sternothyroid, omohyoid and sternocleidomastoid muscles; the internal jugular vein and the deep cervical lymph nodes and the submaxillary triangle and its contents. If a bilateral dissection is done, the jugular vein is not removed on the least affected side. A tracheotomy is performed at the completion of the operation. This is especially important in order to maintain adequate air-way during the course of treatment of deep irradiation therapy.

Complications: In the dissection, some small branches of the cervicomandibular nerve may be injured causing a weakness of some of the muscles of the lower face. The spinal accessory nerve may be severed, causing a handicap in elevating the shoulder on that side. The thoracic duct may be injured in the low dissection, and if so, the duct should be ligated.

Of three patients in the high malignancy group, a decompression and a tracheotomy were performed on only one, the other two refusing any operation whatsoever.

The ten cases here reported fall into the classifications of Ward and Hendrick,[7] as follows:

Low Grade Malignancy:
    Adenocarcinoma   1
    Papillary cystadenocarcinoma   3

Moderate Grade Malignancy:
    Hurthle cell   2
    Lateral aberrent   1

High Grade Malignancy:
    Giant cell   1
    Diffuse type   2

REPORT OF CASES

Low grade malignancy: A female patient 29 years of age first noticed a lump in her neck April 17, 1953. The patient consulted me on June 23, 1953, and examination showed a large lymph node, freely movable, beneath the right sternomastoid muscle. No other lymph nodes were palpable. The thyroid was not enlarged, and no thyroid symptoms were present. There was no evidence of any malignant growth in the upper respiratory tract. Aspiration biopsy was advised after thorough examination, but the patient decided she would wait until Fall before having anything done. On September 16th, 1953, aspiration biopsy of this lymph node revealed a papillary adenocarcinoma.

On September 28, 1953, neck dissection on the right side was done, with removal of muscles, node and the jugular vein (as described above under "neck dissection") with a total right-sided thyroidectomy. The only evidence of malignancy in the thyroid was on the posteromesial surface of the gland where it was attached to the trachea. Histologically this area was a papillary cystadenocarcinoma. The patient has done well to the present date.

The second patient, a male, 54 years of age, consulted me on October 24, 1951, with a lymph node in the suprasternal notch, and one over the submaxillary gland on the left side of the neck. A biopsy of the node in the suprasternal notch proved it to be a papillary cystadenocarcinoma. A neck dissection with the isthmus, and left lobectomy plus the submaxillary gland, and its lymph nodes, was carried out. The patient is alive and well as of February 8, 1955, with no recurrence.

The third patient, a female, aged 59 years, I saw in consultation January 16, 1952 with enlargement of the right lobe of the thyroid with lymph node enlargement on the same side; also a cystic mass just above the thyroid notch attached to the hyoid bone. On January 17, 1952, a neck dissection was done, with a total lobectomy on the right side, and a partial lobectomy on the left; the hyoid bone with cyst was removed en masse; this was followed by a tracheotomy. The histological report was papillary cystadenocarcinoma of the thyroid with metastasis in the lymph nodes. The patient is alive and well three years following the operative procedure.

The fourth patient a 70 year old female, came to my office January 24, 1950. Examination showed the thyroid large and very firmly attached and immovable, with a few large nodes in the upper

part of the thyroid. The patient was very nervous, with hypertension and loss of weight. She would only consent to a biopsy of one node; this was done on February 14, 1950, and the histological report was, adenocarcinoma of the thyroid. The patient refused any operation, as well as any irradiation therapy; she died one year and eight months later.

There is no telling how long this patient might have lived if operation had been performed, as there were no palpable nodes in the neck and this was a low-grade malignancy.

Moderate grade malignancy: A male, four years of age first came to my office March 4, 1949 with a history of having a respiratory wheeze, when excited, for the past three months. Patient had a firm lymph node on the left side of the neck along the deep cervical chain, and another lymph node in the pretracheal area. This pretracheal lymph node, removed for histological study, proved to be a papillary adenocarcinoma of an aberrent lateral thyroid.

On March 7, 1949, a neck resection with a left total thyroidectomy was performed on the left side. The firm mass was resting on the carotid sheath; the carotid as well as the bifurcation was firmly adherent to, and incorporated with the carotid vessels, and could not be freed from them. To successfully remove this it would have been necessary to remove the vessels which would have caused marked changes in the cerebral circulation. The neck dissection was completed, dissecting as much of the mass from the carotid vessels as possible. A tracheotomy was performed at the end of the operation to safeguard the patient during the period of deep irradiation therapy which was carried out. At the present time, February 1955, six years following operation, the patient is well and without any evidence of recurrence.

All histological sections in this case were reviewed by Dr. Harrison S. Martland as well as the Jefferson Laboratory, and they concurred in the diagnosis of adenocarcinoma, lateral aberrent thyroid.

The sixth patient, a female aged 56 years had been operated upon, on September 21, 1951 for adenoma of the thyroid. Her first visit to me was on January 13, 1953, at which time she had an enlarged lymph node on the left side of the thyroid area. This node was removed January 29, and the histological report was, lymph node regional metastasis of a Hurthle cell tumor of the thyroid. This patient died of a coronary thrombosis six months later. She refused operation.

The seventh patient, a female 39 years of age came to see me November 2, 1945, with a history of having had a thyroidectomy in 1934 in another city, this was followed by irradiation therapy.

At the time of her visit to me she had a bilateral abductor paralysis; a tracheotomy was suggested, but refused until January 10, 1949 when it was done. This is a very long history extending over a period of nine years, the patient having had various opinions. On June 12, 1950, I removed two small lymph nodes in the supraclavicular notch for histological study, and reported Hurthle cell carcinoma of the thyroid. At this time I did not think the growth was operable since it involved her larynx and esophagus. She returned to her home in Ohio where she had additional irradiation therapy. I saw her again on November 30, 1951, and my opinion was the same as to the advisability of operation. Then I heard she was operated on February 4, 1954 in another hospital on account of increasing dysphagia, at which time the operation consisted of ligation of the right common carotid artery, and removal of the right cervical esophagus, pharynx, hyoid and larynx. The final histological report was, recurrent Hurthle cell carcinoma of the thyroid with abundant venous invasion and extension into the carotid artery, thyroid cartilage, larynx, esophagus, pyriform sinus and right sternocleidomastoid muscle. The medical complications were derangement of thyroid and parathyroid metabolism. On March 13, 1954 I further heard that x-rays of the chest showed possibility of metastasis in the left mid-lung field. Nothing further has been heard since, and presumably she has died.

High grade malignancy: The eighth patient, a female, 50 years of age, consulted me for dysphagia on April 23, 1954. She had been unable to swallow solids for the past six or seven weeks; also she had a hard swelling on the right side of the neck in the superior portion of the anterior triangle. Her thyroid was immovable and fixed by diffuse swelling on both sides of the neck. The patient was admitted to the hospital May 5, 1954, and a small lymph node at the upper angle of the anterior triangle of the neck was removed for histological study. The report was, giant cell carcinoma of the thyroid. The patient refused any operation, even a decompression. Deep irradiation therapy was given. Her dysphagia became much worse, the pain almost unbearable, and on May 23, 1954, it was necessary to do an immediate tracheotomy for the urgent dyspnea. The thyroid which was firm and fixed, rapidly enlarged but the patient still refused operation, and she died June 30, 1954.

We had two other patients with giant cell carcinoma of the thyroid, both refusing any type of operation, and both died within a very short time.

SUMMARY

A group of ten cases of thyroid carcinoma, taken at random from my files, is presented, with notes on classification, operative procedures and postoperative deep irradiation therapy.

Invasion of contiguous structures, lymph nodes of the deep cervical chain, occurs in malignancy of the thyroid.

Complete neck dissection should be done in all cases of the papillary type of malignancy of the thyroid.

There was no operative mortality in the patients on which neck dissection was performed.

In the more advanced cases in which the thyroid is firmly fixed to the contiguous structures and diffuse swelling is present, only palliative treatment is possible, usually a tracheotomy for the relief of dyspnea.

224 DELAVAN AVENUE.

REFERENCES

1. Trotter, Wilfred: Purvis Oration on Surgery of Malignant Disease of the Pharynx, British M. J. 1:269 (Feb. 13) 1926; A Method of Lateral Pharyngotomy for Exposure of Large Growth in the Epilaryngeal Region, J. Rhin. Otol. and Laryng. 35:289 (Oct.) 1920.

2. Orton, H. B.: Lateral Transthyroid Pharyngotomy, Arch. Otolaryng. 12: 320-332 (Sept.) 1930.

3. Orton, H. B.: Treatment of Extensive Carcinoma of the Larynx, Laryngoscope 61:488-529 (June) 1951.

4. Orton, H. B.: Evaluation of Radical Neck Dissection (Symposium on Carcinoma of the Larynx), Acad. of Ophth. and Otolaryng. (Oct. 12) 1953, Chicago, Illinois.

5. Martin, Hays and Morfit, Mason: Cervical Lymph Node Metastasis as the First Symptom of Cancer, N. Y. Jour. Surg., Gyn. and Obst. 78:133-159 (Feb.) 1944.

6. DeCourcy, Joseph: Pathology and Surgery of Thyroid Disease, p. 74, Charles C. Thomas, Springfield, Ill., 1949.

7. Ward, Grant E. and Hendrick, James W.: Tumors of the Head and Neck, The Williams & Wilkins Company, Baltimore, 1950, p. 655, Table 41.

XXV

THE CARTILAGINOUS STRUCTURES OF THE NOSE

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Harris P. Mosher, beloved surgeon-anatomist and teacher, left a lasting impression upon his students—the urge to acquire a basic knowledge of the anatomy and physiology of the structures submitted to operative surgery. During his yearly course in anatomy at Harvard Medical School, he pointed out the relatively small amount of available information concerning the anatomy and physiology of the external nose.

The cartilages of the nose, quadrangular septal cartilage, lateral and alar cartilages, subjected to movements by the nasal musculature, play an important role in nasal physiology. Their function is inhibited, for example, in facial paralysis, the cartilages of the nose being immobile due to paralysis of the musculature. An inadequate nasal airway is noted on the paralyzed side (Fig. 1).

In plastic surgical operations upon the external nose, it is important to remember that the nose is not a static structure to be shaped at will; due consideration must be given to the preservation of the dynamics of the nasal cartilages. A working knowledge of the anatomy of these hyaline structures is a requisite in surgery of the nose.

The cartilaginous portion of the external nose is situated anteriorly to the pyriform aperture (Fig. 2). Limited above by the lower borders of the nasal bones and the frontal processes of the maxilla, the pyriform aperture is bounded laterally by sharp margins. These margins are thickened below and curve medialward toward the anterior nasal spine of the maxilla.

The septal cartilage is a wide quadrangular lamina forming the framework of the anterior-inferior portion of the septum; it protrudes on the face in front of the pyriform aperture. The anterosuperior angle of the septal cartilage, an important surgical landmark, is designated in our clinic as the *septal angle* (Fig. 2). This

Fig. 1.—Illustrating the immobility of the nasal cartilages when the nasal muscles are paralyzed. A. The nose during inspiration. Note the opening of the naris on the unaffected left side. On the right, due to paralysis, immobility of the naris results in obstruction of the nasal airway. B. Appearance of the nares during expiration.

angle is situated immediately above the alar cartilages in an area referred to as the *supra-tip area*. The major portion of the septal cartilage is firmly bound to the vomer, the perichondrium of the cartilage being continuous with the periosteum of the vomer. The lower part of the septal cartilage extends anteriorly over the smooth surface of the nasal spine. The septal cartilage is mobile in this area, side to side movements being possible because of the flexible relations of the cartilage and the bony surface. The perichondrium of the septal cartilage extends outwardly to join with the periosteum of the wider nasal spine, thus forming a type of joint capsule within which lateral movements of the septal cartilage are possible. The mobility of this portion of the septum, known as the *mobile septum*, increases the natural flexibility of the septal cartilage. The free margin of the septal cartilage is separated from the columella by the juxtaposition of two mucocutaneous flaps in the area known as the *membranous septum*. The layers of the membranous septum extend forward, diverging to join with the cutaneous covering of the medial crura which form the cartilaginous support of the columella.

The lateral cartilages are paired structures attached to the medial portion of the frontal process of the maxilla and the nasal bones above,
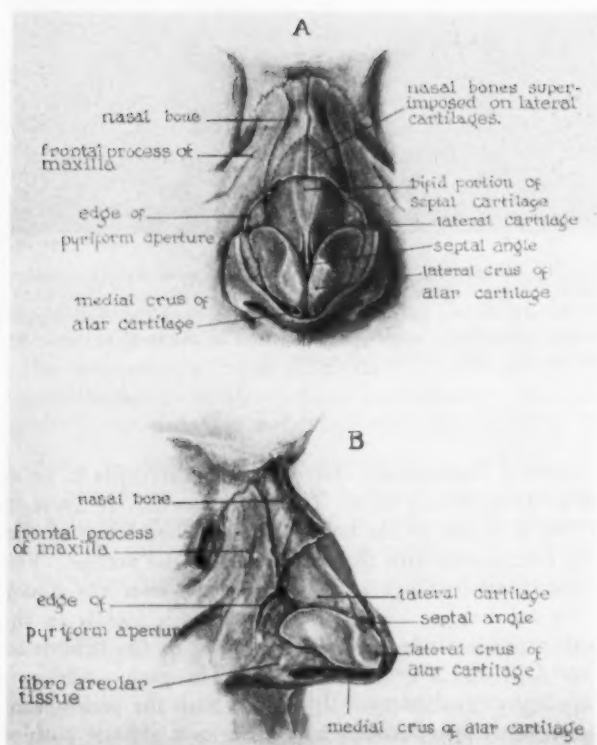
Fig. 2.—Anatomy of the nasal framework.

and to the septal cartilage in the midline. The lateral cartilages are connected below to the alar cartilages by means of dense connective tissue, the upper edge of the alar cartilage overlapping the lower aspect of the lateral cartilage. The outer margin of the lateral cartilage is separated from the edge of the pyriform aperture by fibroareolar tissue.

RELATIONSHIP OF LATERAL CARTILAGES TO NASAL BONES

The relationship of lateral cartilages to nasal bone can be traced to the embryological development of these structures. Because the nasal bones develop from membrane on the surface of the cartilaginous nasal capsule, the lateral cartilage extends upward on the inner surface of the nasal bone. Research now in progress has confirmed the findings of Straatsma and Straatsma.[1] The overriding of the lateral cartilage and nasal bone extends in some instances to 2 cm (Figs. 2 and 3). The fusion of the perichondrium and the periosteum forms an intimate relationship between cartilage and bone. The extent of this anatomical relationship may be observed in a dissection of the nose. When the overlying nasal bone is removed with a small chisel, the upper limit of the lateral cartilage is exposed. This intimate relationship has an important clinical significance, for the lateral cartilage participates in displacement of the bone and is rarely detached from it.

RELATIONSHIP OF LATERAL CARTILAGES TO SEPTUM

The anatomy of the septal cartilage offers a number of points of interest to the surgeon. The septal cartilage also extends upward beneath the nasal bones for a distance equivalent to that of the lateral cartilages. In the region of the septal angle and the adjacent portion of the dorsal border, the septal cartilage terminates in a sharp edge (Fig. 2). The septal cartilage becomes thicker near its junction with the perpendicular plate of the ethmoid; the increased thickness extends to the dorsal border of the cartilage in this area. The dorsal border of the cartilaginous nasal septum undergoes alteration in width and configuration toward the nasal bones. The dorsal border bifurcates into a Y, resulting in a *supraseptal groove* lying between the limbs of the Y (Fig. 3). The groove is readily seen and palpated in some individuals, but it is usually not distinguishable on the surface, being masked by the perichondrium, thick connective tissue, aponeurosis of the nasalis muscle, and subcutaneous tissue. The supraseptal groove is wide near the junction with the nasal bones and is narrower toward the septal angle.
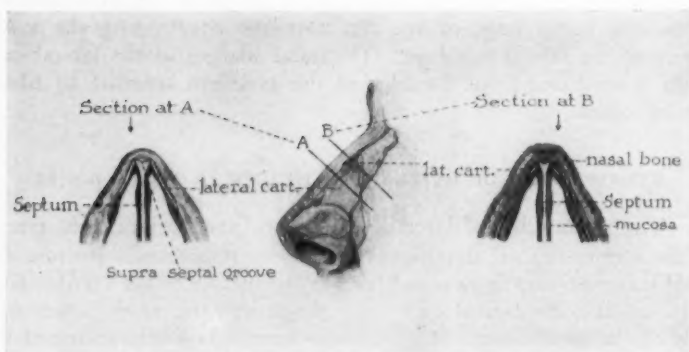
Fig. 3.—Diagrams made from serial sections of the external nose through a frontal plane. The center drawing illustrates the levels *A* and *B* at which sections have been made. Sectional view *A* demonstrates the relationship of lateral and septal cartilages. Sectional view *B* shows the relationship of lateral and septal cartilages with the nasal bones.

The nasal hump, a widened, often prominent portion of the dorsum, is formed above by the nasal bones and below by the widened portion of the septal cartilage. The dorsal hump is fusiform, narrow above, wide near the junction of lateral cartilages and nasal bones, and again narrow above the septal angle.

The lateral and septal cartilages to do not overlap, but join end to end. The structures are intimately connected near the nasal bones, to such an extent in some instances, that there seems to be cartilaginous continuity. Histological examination, however, always reveals a separation between the ends, although a continuity of the perichondrium is frequently observed (Fig. 3). Dense connective tissue binds the cartilaginous ends so closely that the intimacy of the continuity is very great. The two structures are separated by a narrow cleft which becomes obvious toward the septal angle. Fibroareolar tissue in this area permits inward and outward movements which are important functionally. This is the area of the *internal naris* which plays an important role in the passage of air from the vestibule to the nasal fossa proper.

THE ALAR CARTILAGES

The alar cartilages are paired structures which form the cartilaginous framework of the tip of the nose (Fig. 2). Each alar

cartilage consists of two portions, a medial crus and a lateral crus, joined at an area called the *dome* of the alar cartilage, highest point of the tip of the nose. The medial crura, after their junction, curve medially and downward to the columella and are rotated, the upper surface becoming medial. They diverge as they extend downward, the maximal divergence being reached at the widened base of the columella. While there are variations in the size and shape of the alar cartilages, the lateral crus usually occupies little more than the medial half of the ala. The remaining portion of the ala is supported by fibroareolar tissue characteristic of the nose. The border of the nostril is supported by collagenous tissue arranged in longitudinal bundles, supplying a characteristic resiliency.

The alar cartilage is intimately joined to the skin in the columella, where the medial crus is dissected from the skin only with difficulty. The lateral crus approximates the free margin of the external naris border in its medial third, and extends away from the border of the nostril. This anatomical conformation governs the direction of a marginal incision, for one should recall that the free margin extends farther away from the nostril border in its lateral portion. Also, in dividing the alar cartilage into upper and lower segments, the incision should follow the curve of the free margin of the cartilage in order to avoid excising all of the lateral portion of the cartilage.

The dome, point of union of lateral and medial crura, is separated from the margin of the nostril by a triangular shaped area, the *soft triangle,* which consists of two juxtaposed layers of the skin, the covering skin of the nose, and the lining vestibular skin, separated by loose areolar tissue (Fig. 4). An incision close to the nostril border should be avoided; when exposing the dome, the free margin of the alar cartilage must be followed, thus avoiding incising through the soft triangle. Postoperative notching of the nostril border is thus prevented.

The lateral crura of the alar cartilages diverge one from the other in the supra-tip area, leaving between them a triangular-shaped area into which is fitted the septal angle. Because the dorsum in this area is supported only by the septal angle, it is referred to as the *weak triangle* (Fig. 5). Lateral and alar cartilages are connected by aponeurotic-like tissue which also maintains the alar cartilages attached to the septal angle, thus acting as a suspensory ligament of the tip of the nose. The tip of the nose droops when the support of the septal angle is lost by injury or operative removal. This deformity may be accompanied by a retraction of the columella which is also due to loss of support by the septal framework.
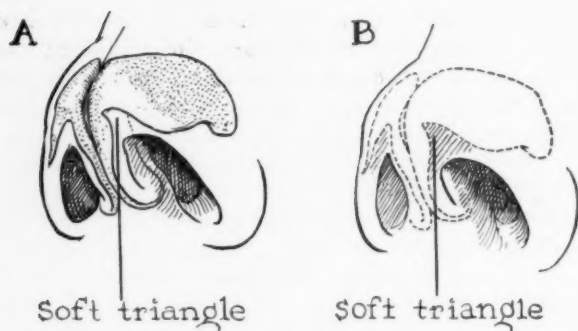
Fig. 4.—The soft triangle. *A.* A triangular-shaped area consisting of two juxtaposed layers of skin separates the dome of the alar cartilage from the nostril border. *B.* The soft triangle is represented by the shaded area.
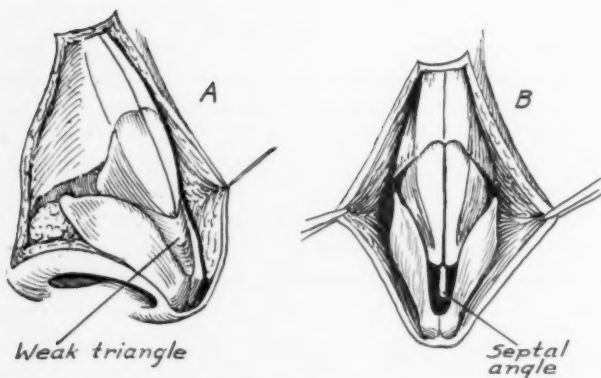
Fig. 5.—The weak triangle. *A.* Dissection showing structures after the skin, subcutaneous tissue and muscular layer are raised. The aponeurosis covers the triangle between the alar and lateral cartilages, over the septal angle. *B.* Septal angle exposed after removal of the aponeurosis. (From Converse, J. M.: Corrective Surgery of Nasal Deviations, Arch. Otolaryng. 52:671, 1950.)

THE COLUMELLA

The columella extends from the tip of the nose to the lip, joining the latter at the upper portion of the philtrum and separating the external nares. The posterior portion of the columella is wider than the anterior portion, due to the divergence of the medial crura of the alar cartilages, the lower ends of which embrace the lower margin of the septal cartilage. The contour of the columella depends essentially upon the shape and the degree of flare of the medial crura of the alar cartilages. The widening at the base of the columella is due to the outward flare of the lower ends of the medial crura.

THE VESTIBULE

The vestibule extends below the floor of the nose as is evidenced when the palpating finger encounters the crest which delimits the lower portion of the pyriform aperture. The lower border of the lateral cartilage protrudes into the vestibule forming a fold, the limen nasi or *internal naris* (Fig. 6). This fold is prolonged downward and medially along the crest of the pyriform aperture. The inspired air is filtered, warmed and moistened in the vestibule. The valve-like action of the internal naris, through variations in position of the lateral cartilage in relation to the septum, aids in controlling the current of air which enters the nose.

Nasal ventilation depends upon the efficiency of the lateral and alar cartilages: active motion by contraction of the nasal musculature, and passive movements due to variations of intranasal pressure. The connective tissue which binds lateral, alar and septal cartilages permits movements imparted by the external nasal musculature. Interference with the function of the external nose may result from faulty surgical procedures. These include destruction of the part of the nasal musculature in an effort to thin the nasal structures; excessive removal of cartilage, and consequently of the framework upon which the nasal musculature acts; too wide resection of cartilage and vestibular lining resulting in stenosis at the internal naris; operative procedures producing rigidity in the mobile and membranous septum and columella by overshortening the septal cartilage; excision of the membranous septum, or injudicious implantation of cartilage between the layers of the membranous septum. It is essential to preserve the function of the depressor septi nasi muscle for this structure tenses the membranous septum as nasal inspiration begins (Fig. 7).

SUMMARY

The anatomy of the cartilaginous structures of the nose is reviewed and a practical terminology suggested for some of the struc-
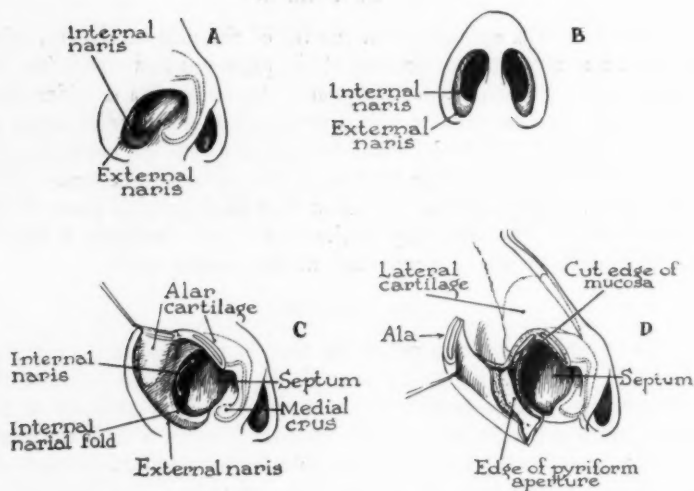
Fig. 6.—The internal naris. A and B. The external nares, limits of the nasal vestibule. C. The ala has been sectioned in order to show the internal naris, formed by the protruding lower border of the lateral cartilage, prolonged downward by the internal narial fold. D. The edge of the pyriform aperture is exposed demonstrating that the internal narial fold extends along the crest of the pyriform aperture.
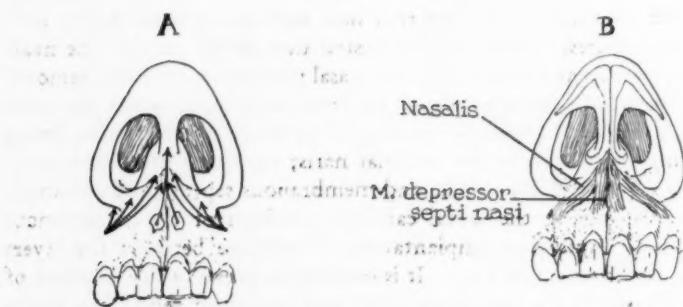
Fig. 7.—The depressor septi nasi muscle. A. Diagram showing the direction of the pull of the depressor septi nasi muscle. B. Drawing representing the depressor septi nasi muscle joined by fibers from the nasalis penetrating through the base of the columella.

tures discussed.   The important role of these cartilages in the physiology of the external nose is emphasized in relation to surgical procedures.

550 FIRST AVENUE.

REFERENCE

1. Straatsma, B. R. and Straatsma, C. R.: The Anatomical Relationship of the Lateral Nasal Cartilage to the Nasal Bone and Cartilaginous Septum, Plastic and Reconstructive Surgery 8:443, 1951.

XXVI

PITFALLS IN THE TREATMENT OF OTOLARYNGO-
LOGICAL CONDITIONS

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CLAYTON, MO.

Whether one would wish to designate the present status of the specialty of otolaryngology as a "depression" or as a "recession," in consonance with the modern idea of expressions applied to economic trends, it is clearly apparent that surgery of the paranasal sinuses and the mastoid for the relief of infections has been definitely curtailed. While the reason for this has been attributed to the widespread use of the sulfonamides and the antibiotics, other factors have also had an important influence.

In the pre-sulfonamide-antibiotic era we have seen the treatment of sinusitis change either to the radical or the conservative in one locality or another as a result of the influence of those who held the respect of their fellow otolaryngologists and have thus shaped the policy, however unconsciously, of the conduct of their associates, by reason of their stronger medical or surgical inclinations, respectively, towards otolaryngologic problems. Thus radicalism and conservatism wisely or otherwise, in the treatment of sinus infections, has followed an ebb-and-flow pattern. The pattern of infections, which may change from year to year or cyclically, is likewise an important factor of which we are fully aware.

These variable endemic episodes are as frequently the result of the occurrence of different strains as of entirely different bacteria, or of viruses which show variations in resistance to therapeutic agents, especially since the advent of chemotherapy. The lack of recognition of these factors may often lead to false and unwarranted modifications of therapeutic methods.

This basic trend no doubt still exists. However, it has been modified by the more accurate diagnostic methods which have been developed and by our greater knowledge of nasal physiology and of nasal allergy.

The works of Proetz,[1] Hilding[2] and others have justifiably alerted the otolaryngologist to the importance of the physiology of the nose and the paranasal sinuses. They have stressed the exact functions of the cilia and of the mucosa of these areas, and have furthermore warned us of the injurious effects of injudicious intranasal medicaments upon the normal physiological functions of the mucosa. We have consequently become well aware of the irreparable interference with these functions which follows the removal of turbinates and sinus walls in radical surgery. We have thus been enabled to distinguish between disturbed or suspended physiology and irreversible pathology of the mucosa of the nose and paranasal sinuses.

The otolaryngologist is now more conscious than ever of the significance of pathological changes and has a greater respect for the preservation of nasal and sinus physiology, and of hearing, leading to a conservatism of his own choosing, not one wholly governed by force of circumstances.

As a specific example, I may cite a reconsideration from a clinical standpoint of my report[3] on the pathological changes observed in the mucosa of the turbinates and sinuses of 70 patients operated upon for sphenoethmoidal sinusitis by Dr. Greenfield Sluder and some members of his staff at Barnes Hospital, Washington University School of Medicine, immediately prior to 1926.

These patients were operated upon to relieve a variety of symptoms, namely, headaches in various locations, sneezing, coryza, nasal obstructions (transient or constant), nasal discharges (purulent, mucoid and watery), asthma (seasonal and perennial), arthritis, pulmonary abscess, visual disturbances or actual blindness, nephritis and anorexia.

Some, or all of the above clinical symptoms were either moderately or entirely relieved in 70 per cent (49) of these patients, with little or no relief in 30 per cent (21) patients. The symptoms had been present from one to thirty years preoperatively, and the above results annotated after 1-18 months postoperatively. Sixteen of the total number (22.8 per cent) gave a history of symptoms indicative of allergic rhinitis, as so construed to-day, and 12 of these patients (24.5 per cent) were among the 49 who had obtained appreciable relief from their surgery. Assuming that these twelve, and possibly the entire sixteen allergic patients, would benefit equally well and probably better as the result of our more advanced knowledge of allergic diagnosis and therapy, if they were not subjected to surgery with the removal of physiologically important tissues in which reversible pathological changes are present, we may acknowledge a

voluntary reduction by the otolaryngologist of 22 per cent to 25 per cent of this type of sinus surgery.

Fully realizing that deductions only may be drawn from such a small number of cases, these figures may serve to corroborate one of the reasons for the reduced amount of sinus surgery being performed at the discretion of the otolaryngologist. Nevertheless, it should be the function of the otolaryngologist to decide which 25 per cent of patients with symptoms of sinusitis, from infection or allergy should not be operated upon as judged by the intranasal pathology and findings, the x-ray changes found in the paranasal sinuses, and the general condition of the patient. Too often the internist, the pediatrician or the general practitioner will decide that a sinus infection is, or is not of pathognomonic importance on the x-ray report alone, and misjudge or ignore the intranasal pathology.

ALLERGY

Hansel[4] and others have stressed the importance of skin testing and of elimination diets to determine sensitiveness to the various offending substances with which the individual may be coming into contact. The information thus obtained may be used to guide the patient in his efforts to be relieved of his allergic symptoms. Likewise, desensitization with specific allergens has proved useful in giving further relief.

IONIZATION

Among the many treatments resorted to in order to attempt to relieve the symptoms of seasonal allergic rhinitis, ionization of the nasal mucosa was probably the most drastic. Certain otolaryngologists[5] enthusiastically reported a high percentage of good results, and even cures of patients who had suffered from ragweed, and at times, perennial allergy for many years.

In 1933-1935 we conducted experiments on dogs in which the nose and frontal sinuses were ionized with 2 per cent zinc sulphate solution, and the microscopic changes in the ionized mucosa and bone were studied after the dogs were sacrificed in 18 hours, 2, 4, 7, and 12 weeks.[6] Our conclusions were:

"1) Definite destructive changes were brought about in the mucosa of the frontal sinuses of dogs by the ionization of the zinc sulphate solution. These changes consisted of ballooning, fragmentation and complete destruction of the surface epithelium, a marked edema of the subepithelial tissues and an extravasation of free red blood cells into these tissues from greatly dilated and ruptured capillaries.

"2) Ionization of the frontal sinuses caused a fibrosis of the mucosa in dogs killed at seven and twelve weeks, respectively; and marked hyperplastic changes in the bone (hyperostosis) in the dog killed at twelve weeks.

"3) Ionization of the noses of dogs with a solution of zinc sulphate caused a fibrosis of the subepithelial tissues which was seen to be progressively more marked and well established in the animals killed at intervals of from eighteen hours to seven weeks following ionization."

From these findings it was apparent that since generalized destruction of the nasal and sinus mucosa resulted from ionization, the subsequent fibrosis was highly undesirable and unjustifiable. Increased and excessive fibrosis means depletion of the blood supply to these structures and further fibrosis; it means atrophy of the glands due to compression, causing repression of secretions, and compression of nerves, with the imminent probability of giving rise to nasal pains, hyperesthesia, neuritis and headache of a more or less intractable nature, even though there was apparently no damage to the nerve structures themselves.

Shortly thereafter this method of treatment was abandoned, since it was not reasonable to establish a state of dysfunction in a nose for ten months of the year in order to achieve questionable and diminishing relief from symptoms of ragweed allergy for the remaining two months.

ANTIHISTAMINICS

By the same token the antihistaminics are variously misused. These unquestionably serve a valuable purpose in the treatment of urticarias from sensitivities resulting from certain drugs, particularly the antibiotics, when combined with the withdrawal of the offending medication; in angioneurotic adema, for the relief of the nasal symptoms of allergy, viz. sneezing, rhinorrhea and obstructed airway, as a temporary adjuvant while the basic allergic diathesis is being properly diagnosed and treated, and in the early stages of an acute idiopathic rhinorrhea, for symptomatic relief. However, these drugs when taken beyond this early stage serve to increase the viscosity of the ensuing purulent discharge, and thus by imposing too great a burden upon the partially dried cilia within the sinuses and the nose interfere with the proper and orderly extrusion of the secretions and eventually lead to an insurmountable accumulation of infected secretions within these cavities and spaces, with subsequent aggravation of serious local and constitutional symptoms. Yet, they are prescribed and permitted to be taken for indefinite periods of time to

bring about a diminution of nasal discharge regardless of type or character.

We have seen many patients in this category for whom it was necessary to irrigate the maxillary sinuses and evacuate large amounts of semidried, tenacious pus which resulted from dehydration of the secretions after the prolonged use of antihistaminic medication, especially in conjunction with the antibiotics, to counteract possible sensitivities.

The antihistaminics, nevertheless, have a place in the treatment of some cases of allergic rhinitis, allergic eustachian salpingitis and for secretory otitis media, idiopathic labyrinthitis, edema of the vocal cords and for the relief of the symptoms of certain asthmatic conditions.

The undesirable side effects of the antihistaminic drugs are so well known that comment would be superfluous. Some are stimulating to the patient, and others cause depression or drowsiness thus, careful study and consideration should be given of the type of patient for whom they are prescribed.

ANTIBIOTICS

Since the discovery of penicillin by Fleming[7] in 1929, the medical and lay world has become increasingly "antibiotic conscious." As stated in "Antibiotic Therapy"[8] the term *antibiosis*, meaning "the survival of the fittest," probably was first used by Vuillemin[9] in 1889. About ten years later Marshall Ward used the term "antibiosis" to describe microbial antagonisms, and in 1942 Waksman[10] proposed the use of the term "antibiotics" to define those clinical substances of microbial origin which possess antimicrobial activity, and this meaning was later enlarged to include chemical substances of plant and animal tissues.

With the advent of penicillin and the subsequent elaboration of the "mycins," the sulfonamide derivatives—sulfonilamide, sulfathiozale, sulfapyridine, sulfadiazene and sulfamerizine—were relegated to a secondary position in therapeutics because of their unfavorable systemic reactions and their narrower scope of application. This, despite the more accurate methods that were routinely used to corroborate the optimum blood content of these drugs (12-15 mgm) as an index of the proper dosage. This is in marked contrast to our present methods of administering the antibiotics, where simple methods for the determination of blood content are not available.

Possibly there would be fewer toxic side reactions and fewer unnecessary sensitizations of patients, if less complicated and more

rapid methods were worked out to determine the sensitivity of the specific infecting micro-organisms to the individual antibiotic before it is administered to the patient, and subsequent evaluations of the optimum blood concentrations carried out. Probably the reason lies in the fact that the patient is usually ambulatory and the sensitivity tests so time-consuming that they are not used unless under critical circumstances.

Realizing the importance of the time element in the currently used methods of determining the sensitivities of micro-organisms to the various antibiotics, a matter of 48 to 72 hours before a report is obtainable from the laboratory, we have experimentally confirmed the degree of accuracy of a 24-hour method as compared with the 72-hour method.[11]

In essence, this consisted of flooding blood agar plates with a nutrient broth which had been inoculated with a swab culture taken from the focus of infection in the nose, throat or ear of the patient, representing the entire bacterial flora. The discs containing specific amounts of the various antibiotics were placed on the inoculated plates which were incubated for 24 hours and then read. The compilation of the results from this method revealed that it was 80 per cent as accurate as the 72-hour method, with the added advantage of a positive report in one third the time, and a definite saving of material and technicians' time. Nevertheless, these advantages can only be realized as a routine measure if the patients are hospitalized or being treated in the out-patient clinic, since adequate laboratory facilities are not readily available in ordinary office practices. We must therefore await simpler methods for such determinations.

Withal, may we not be too precipitate in inaugurating the administration of the antibiotics, lest we deprive the patient of the benefits to be gained by exposure to pathogens a sufficient length of time to enable the body mechanism to elaborate antibodies sufficiently to cope with the specific micro-organisms?

In our anxiety to accomplish a cure of an infection as rapidly as possible by the early administration of antibiotics, we may lose sight of the fact that it is the resistance of the individual, the action of the immune bodies, the phagocytes, which eventually cure the patient, since the function of the antibiotics is bacteriostatic and rarely bactericidal. From our knowledge of immunology, principally from the tenets of Ehrlich and Pasteur, we know that immunity is natural and acquired, or both, in the human species, and that the formation of specific antibodies is stimulated by the presence of tissue infection. If therefore, infection is greatly attenuated before such stimulation

can be attained and antibodies elaborated, the resultant condition of the patient is one of greatly lowered resistance to the present and to subsequent similar infections.

The lay public has conceived the idea that antibiotics are truly "wonder" drugs; that all that is necessary to control and quickly cure any respiratory infection is a 'shot' or two of one of other of these drugs, which they frequently insist upon, and they can then pursue their usual activities, completely ignoring the cardinal principles of good patient care, viz. proper rest, proper elimination, and adequate fluid and food intake. In a way, the physician is responsible for this attitude of false security in not explaining to the patient that these cardinal principles must be adhered to in order to enable him to build up his resistance to combat infection which the antibiotic is intended to control.

It is undeniable that antibiotics are frequently administered too readily and too early in acute infections. The danger of sensitization of a patient is ever present and must be faced, but with the realization that the end must justify the means. It is a lamentable tragedy to deprive a patient of the benefit of an antibiotic in a subsequent serious illness because he has been sensitized at the time of a simple infection which would have responded to ordinary therapeutic measures or have been self-limited by the innate resistance of the individual. These factors should be determined by the experience and diagnostic acumen of the physician. This diagnostic acumen must be modified and tempered by the realization that the use of antibiotics has not only changed the so-called "textbook picture" of the symptomatology and the signs of infections, but has likewise influenced the virulence of micro-organisms and increased their resistance to specific antibiotics.[12] This latter factor may be actually exemplified by the transmission of such antibiotic resistant micro-organisms, the virulence of which has been greatly enhanced by animal passage, from one individual to the other, thus establishing an endless chain of subsequent infections, conceivably of epidemic proportions.

The warning cannot be too often repeated of the danger of "blind" treatment with antibiotics of otitis media in children, whether it be secretory, serous or suppurative. The patient with an ear of this type which has become ostensibly asymptomatic, but unresolved, in which a paracentesis is not done, and proper audiometric observation not pursued, is a potential subject for a chronic hyperplastic otitis media, and a later victim of the handicap of an irreversible conductive deafness. This course of action is particularly reprehensible where such facilities are readily available, but not taken advantage of.

TONSILLECTOMY AND ADENOIDECTOMY

Tonsillectomy and adenoidectomy are definitive therapeutic procedures when indicated, and the subsequent benefits to the patient are unquestionable. But, should we deprive the patient of these benefits by abandoning this practice because of the recently aroused apprehension of the possibility of contracting poliomyelitis if the operation is performed during the "polio season" or, because certain statistical reports have created the impression that the tonsillectomized individual is more prone to contract bulbar poliomyelitis? Or, should we permit the children who are afflicted with diseased tonsils and adenoids to remain under-nourished, apathetic and devoid of resistance to infection, or the children who have suffered from repeated attacks of otitis media to become eventually deafened, and not remove the tonsils and adenoids because of the fear that they might contract poliomyelitis at some time or other? Are some of the sequelae of poliomyelitis to be dreaded more than those of acute rheumatic fever? These are only a few of the important questions which we must ask ourselves if we are to be conscientiously consistant in our practice of otolaryngology.

From the foregoing brief resumé of the present status of therapy in otolaryngology, it seems fairly clear that we may be going through a "recession" in the sense that there is a temporary paucity of certain types of clinical material, because we are in a cycle of conservatism or because of the widespread use of the antibiotics, whether judiciously or otherwise, but that this phase will be limited because of the necessary curtailing of the use of these drugs on account of sensitivities in patients, bacterial resistance and modified pathogenicity.

TRAINING IN OTOLARYNGOLOGY

There will thus still be a place for sinus and mastoid surgery and a need for men trained to perform these operative procedures. Laryngeal and neck surgery should remain in the field of otolaryngology, as should plastic and reconstructive surgery of the nose, the paranasal sinuses and the ear. It is highly regrettable that in certain medical schools, the undergraduate curriculum in otolaryngology is being reduced to little more than token "brush off" courses of instruction and training and the assignments to otolaryngology during internship, negligible. It is difficult to reconcile this attitude with the oft repeated lament that students are not properly trained for the general practice of medicine. Is there not a serious injustice being perpetrated against the future physician by the pursuance of this policy?

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SUMMARY

In an effort to analyze the recent trend in the practice of otolaryngology a brief resume of some factors which may have a bearing is presented.

1. There have always occurred waves of radicalism and conservatism in the surgery of the sinuses and ear as the result of the types of infection that may prevail as well as the influence of certain eminent practitioners who may have strong leanings to radicalism or conservatism.

2. Our more accurate knowledge of the physiology of the nose, the paranasal sinuses and the ear, and of the allergies has undoubtedly been a deterrent to injudicious surgery.

3. The widespread use of the antibiotics has contributed largely to the expedient of medical otolaryngology, but it is becoming more apparent that their uses will be increasingly curtailed because of the sensitivities of the population at large and the wider incidence of antibiotic resistant pathogenic micro-organisms.

4. The teaching of otolaryngology and the training of residents should not be neglected since there is a great likelihood that it will be necessary to revert to the basic methods of treating otolaryngological conditions in the future.

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REFERENCES

1. Proetz, A. W.: Essays on the Applied Physiology of the Nose, St. Louis, Mo., The Annals Publishing Co., 1941.

2. Hilding, A.: Direction and Rate of Drainage of Mucus Secretions From the Internal Surfaces of the Nose, Proc. Staff Meet., Mayo Clinic 6:285, 1931.

3. McMahon, B. J.: Pathology of Spheno-Ethmoidal Sinusitis, Arch. of Otolaryng. 4:310-333 (Oct.) 1926.

4. Hansel, F. K.: Clinical Allergy, St. Louis, Mo., The C. V. Mosby Co., 1953.

5. Alden, A. M.: Ionization for the Allergic Patient, Trans. Amer. Laryng. Rhin. and Otol. Soc. 43:323, 1937.

Hurd, L. M.: Ionization Treatment of Hay Fever and Hyperesthetic Rhinitis, Trans. Amer. Laryng. Rhin. and Otol. Soc. 41:109, 1935.

Tobey, H. G.: Ionization Treatment of Nasal Mucous Membrane, Trans. Amer. Laryng. Rhin. and Otol. Soc. 41:302, 1935.

Montgomery, D. C.: Ionization Treatment in Hay Fever, Trans. Amer. Laryng. Rhin. and Otol. Soc. 42:508, 1936.

6. McMahon, B. J.: Effects of Ionization on the Mucosa of Frontal Sinuses of Dogs, Annals of Otology, Rhinology and Laryngology 43:643 (Sept.) 1934.

McMahon, B. J.: Late Changes in the Mucosa of the Frontal Sinuses and Nose of Dogs Following Ionization, Arch. of Otolaryng. 22:454-465 (Oct.) 1935.

7. Fleming, A.: The Antibacterial Action of Cultures of a Penicillium with Special References to Their Use in Isolation of B. Influenzae, Brit. Jour. Exper. Path. 10:226-236 (June) 1929.

8. Welch, H. and Lewis, C. N.: Antibiotic Therapy, Arundel Press, Washington, D. C., 55, 260, 1951.

9. Vuilleman, P.: Antibiose et Symbiose, Assoc. Franc. pour. L'Avance d. sc. Part. 2:525-543, 1889.

10. Waksman, S. A.: Microbial Antagonisms and Antibiotic Substances, The Commonwealth Fund, New York, 1945.

11. McMahon, B. J.: The Antibiotics As an Adjunct in the Treatment of Otolaryngologic Diseases, The Laryngoscope 64:208-217 (Mar.) 1954.

McMahon, B. J.: Seasonal Variations in Sensitivity of Micro-Organisms to Antibiotics, to be published in the Jour. of the South. Med. Assoc.

12. Haight, Thomas and Finland, Maxwell: Resistance of Bacteria to Erythromycin, Proc. Soc. Exper. Biol. and Med. 81:183, 1952.

XXVII

CARDIOSPASM

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Mosher's closing sentences in his remarks "On Being a Professor Emeritus" were, "The strength of specialism is specialism. Paradoxically its weakness also is specialism."[1] These words have particular significance to Mosher's own extremely important contributions to an understanding of diseases of the esophagus. Specialization permitted the development of esophagology as a subspecialty of otolaryngology, and Mosher's investigations and publications in this field helped in maintaining it as an integral part of otolaryngology. The weakness of such superspecialization has been a certain degree of isolation of these contributions; they are well known in the literature and practice of otolaryngology, but often there is a lack of correlation and occasionally a failure of recognition or understanding in surgical and medical fields of the priority and significance of these contributions.

Cardiospasm is one of the commonest esophageal lesions met with in clinical and endoscopic practice. It has been defined as a nonorganic obstruction at the cardioesophageal junction associated with atrophy and atonia of the esophageal walls. In cases of long standing, Mosher[2] has recognized the importance of fibrosis of the terminal portion of the esophagus with disorganization of the musculature or its complete replacement by fibrous tissue. Unfortunately, the controversy over etiology has given rise to a complex nomenclature. The earlier authors have offered a variety of terms, each of which has stressed a particular feature of the disorder. Thus, the anatomic manifestations were stressed by such terms as megaesophagus, fusiform esophagus, simple ectasia and idiopathic dilatation of the esophagus while achalasia of the cardia, cardiospasm and phrenospasm have

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each suggested etiologic factors. The number and variety of terms indicates the lack of agreement in regard to the essential features of the condition.

The purpose of the present report is to review some of the clinical features of cardiospasm demonstrated in a series of 189 cases and to correlate etiologic, medical, endoscopic and surgical phases of the problem.

ETIOLOGY

The terms cardiospasm and achalasia of the cardia are perhaps the most unfortunate in that attention is at once focused on the cardio-esophageal function. The available evidence, however, supports the view that the neuromuscular function of the entire smooth muscle portion is at fault, and in referring to his own series of cases, Mosher[3] stated, "spasm played but a minor part in cases as they came to our hospital." Yet the term "cardiospasm" is so widely accepted, it will probably never be discarded and seems permanently associated with this specific, well recognized clinical entity. In 1949 Kramer and Ingelfinger[4] using balloon kymographic methods demonstrated loss of tonus, lack of propulsion and an irregular disorganized wave pattern in the upper esophagus in individuals with cardiospasm. In addition, the response to parasympathetic stimulation with acetyl-beta-methyl choline was in marked contrast to the increase in tonus exhibited in normal individuals. Templeton[5] in 1948 reported similar abnormalities in esophageal motility by studying the wave patterns seen at fluoroscopy. The failure of the primary peristaltic wave to progress past the aortic arch and the purposeless shallow contractions of the thoracic esophagus also contrasted markedly with the motility pattern seen in other obstructing lesions such as carcinoma. On the other hand, it is important to note that the extreme degrees of esophageal dilatation seen in cardiospasm are rarely encountered in other fibrotic and neoplastic lesions occurring at the cardia.

While the factor or factors responsible for the severe dilatation, loss of tone and other characteristics of this process remain unknown, the weight of experimental evidence favors the view that this lesion is primarily a disease of the myenteric plexus.[6] The neurogenic control of the distal esophageal segment was demonstrated by Knight[7] in a series of balloon kymographic experiments in cats, in whom he was able to reproduce the syndrome of cardiospasm by vagotomising his animals. Sympathectomy (D3 to D10) prevented the development of cardiospasm following vagotomy, while coeliac periarterial sympathectomy could reverse the process once it had become established. He subsequently reported two cases[8] successfully managed
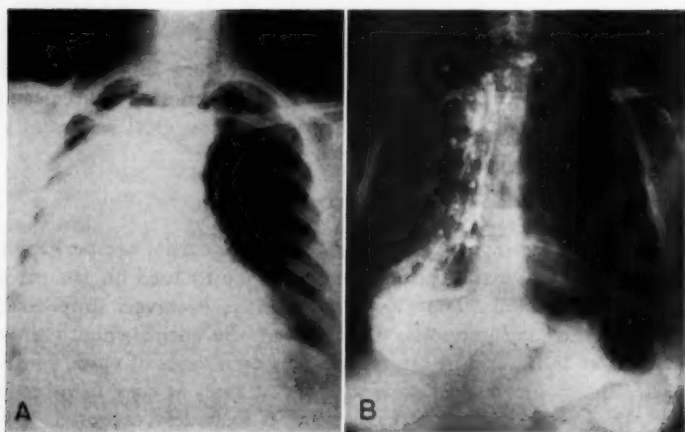
Fig. 1.—Extreme megaesophagus with severe food and fluid retention in a female 43 years of age who remained obese through the duration of symptoms.   A. Flat "scout" chest film.   B. Five hours after barium meal.
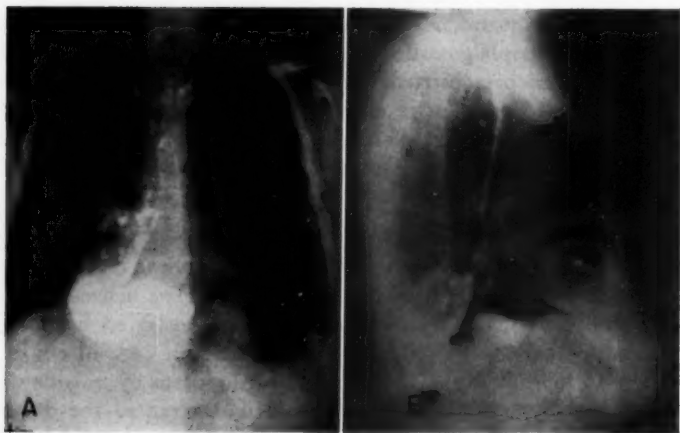
Fig. 2.—Redundant, tortuous esophagus resting on the diaphragm.

by coeliac ganglionectomy in humans. This approach, although physiologically rational, has failed to be of value in clinical application. Improvement seemed subjective, with persistence of obstruction and megaesophagus being apparent on fluoroscopic examination. The variability of response to neurogenic stimulation shown by cats, rabbits and dogs, and even by different animals of the same species under identical experimental conditions accounts for the failure to solve this problem through the use of experimental animals.

From the standpoint of further evidence of the neurogenic etiology of this condition, it is important to record the transient development of the typical clinical picture of cardiospasm in four patients not included in this series following vagotomy for a peptic ulcer. Each had temporary dysphagia, esophageal dilatation and the regurgitation of undigested food. Two required peroral dilatation and responded well. In the other two the relief was more gradual but symptoms finally disappeared spontaneously. This functional change at the cardia following vagotomy has been observed by Friedberg[9] and others and is significant in correlating the vagus nerve with the cardiospasm syndrome.

The mechanical concepts of etiology based on the observation of obstruction at the diaphragmatic pinchcock as expressed by Jackson[10] and findings of obliterating infection in the liver tunnel and terminal esophagus by Mosher[11] are not necessarily at complete variance with these observed neurogenic changes. It is probable that these are additional factors of variable significance at different stages of the condition just as, for example, psychogenic influence or associated gall-bladder disease, or other chronic infections of the upper abdomen have a prominent etiologic influence in other cases.

The role of psychogenic factors in the etiology of cardiospasm has been stressed by some authors and vigorously denied by others. Of the 189 cases herein presented, obvious psychoneurosis or significant psychogenic stress was apparent in 19, 7 males and 12 females. These figures probably err on the low side since no concerted study was made of this important phase of the subject in this series. Where a correlation does exist, the question is often raised as to whether the psychogenic symptoms were the cause or the result of the chronic swallowing difficulty.

The patients in this series ranged in age from 6 days to 83 years. No difference in sex incidence was noted, there being 94 males and 95 females. The age distribution in decades is shown in Table I, the age given being the age at which symptoms were first noted.

TABLE I.

Birth to 10 years	5
10 to 20 years	18
20 to 30 years	27
30 to 40 years	34
40 to 50 years	38
50 to 60 years	34
60 to 70 years	17
70 to 80 years	6
80 to 90 years	1
Age unknown	9
	189

The geographical variation in the incidence of cardiospasm is of considerable interest and may have some etiologic significance. While one may consider the disease fairly common in the United States, where a series of as many as 600 cases have been reported from one clinic,[12] it is apparent that the incidence in South America, particularly Brazil, is very appreciably greater. Sabino Vieira De Freitas,[13] for example, reports his observations in a series of 2136 cases treated during a 16 year period in three local hospitals having a combined bed capacity of 350 beds. The condition was seen in a ratio of nine males to one female, and most frequently in the white race. He states further that while some may attribute the condition to an avitaminosis, the rarity of other syndromes associated with deficiency diseases, such as polyneuritis, pellagra, and Plummer-Vinson syndrome is surprisingly low in this same general district.

CLINICAL FEATURES

The most common and distressing symptom of cardiospasm is dysphagia. At first the gradual onset of dysphagia for solid foods is noticed, and may only consist of the sensation of a momentary delay in the swallowing act. This is usually intermittent initially although occasionally the onset is abrupt and rapidly progressive. Gradually the diet becomes more and more restricted until by the time medical assistance is sought, the patient may be found to have been existing on liquids alone. A variety of sensations result from the delay in esophageal emptying, and these may be described as feelings of retrostrenal and epigastric fullness, pressure, heaviness or vague discomfort. Severe pain usually signifies an associated esophagitis. Many of these individuals learn to literally wash the food

TABLE II.—WEIGHT LOSS IN POUNDS.

0 to 10 lbs.	8 patients
10 to 20 lbs.	10 patients
20 to 30 lbs.	8 patients
30 to 40 lbs.	11 patients
40 to 50 lbs.	2 patients
Over 50 lbs.	8 patients
	47 patients

down by drinking large amounts of water with their meals. This is apparently accomplished by building up sufficient hydrostatic pressure to dilate the cardia. This phenomenon is frequently noted by the radiologist who reports a thin trickle of barium entering the stomach when the barium level in the esophagus reaches a height of eight inches or more above the cardia. As dysphagia becomes more severe and dietary restriction more marked, weight loss becomes apparent. At times this is extreme and with the associated weakness, the cachexia of malignancy may be simulated. Loss of 40 or 50 pounds is by no means uncommon in the neglected case and in such instances avitaminosis may coexist with the general nutritional deficiency. Significant weight loss was noted in about 25 per cent of cases varying from less than 10 to more than 70 pounds as illustrated in Table II. In occasional cases, however, the converse is true. Patients may be seen who are actually obese in spite of a high degree of stenosis and the retention of several quarts of food and fluid (Fig. 1).

As decompensation of the esophagus occurs the capacity of the organ increases and symptoms of esophageal overflow make their appearance. Vomiting or regurgitation of undigested food is particularly distressing and many patients find themselves unable to eat in public. Often because of the recumbent position at night, the pharynx is flooded with the contents of the evening meal. The symptoms of esophageal overflow may overshadow the dysphagia, the attacks of choking or strangling being uppermost in the patient's mind. Chronic aspiration of esophageal contents leads to a variety of pulmonary complications, even sudden death. This was reported in one of Mosher's[3] cases and occurred in one 18 year old boy in the series herein reported. Pulmonary symptoms due to aspiration of esophageal contents have been discussed in texts[14] and other reports of cardiospasm. The present series of cases repeats the experience of previous authors with the exception that no case of lung abscess was

Fig. 3.—"S" shaped distal esophagus in patient with severe dysphagia.
It was impossible to pass a bougie into stomach even with a string guide.

found.   In the one instance mentioned of sudden death following
the aspiration of a large amount of regurgitated undigested food,
autopsy examination showed the tracheobronchial tree filled with
material identical in composition to that recovered from the esoph-
agus.   In this series, five patients have listed chronic cough as a pre-
dominant symptom, three nocturnal cough, five have had chronic
or recurring attacks of pneumonia, three had demonstrable bronchi-
ectasis, and one a persistent atelectasis.   Three deaths have resulted
from the pulmonary complications; as indicated above, one died
suddenly after a meal due to regurgitation and aspiration of the esoph-
geal contents; two died of pneumonia, one a 12 year old boy who
had an extensive, bilateral bronchiectasis associated with a severe
cardiospasm.

DIAGNOSIS

The differential diagnosis of organic from functional obstruc-
tion at the cardioesophageal junction is of utmost importance.   The
classical history of patients with cardiospasm is that of dysphagia and
regurgitation of undigested food, especially when lying down and
during sleep.   Episodes of foreign body obstruction are not infre-
quently observed.   Two distinguishing points in the history are sig-
nificant; first, the symptoms of the patient with cardiospasm fluc-
tuate in degree for a considerable period of time, often influenced by

his emotional tension, whereas the symptoms of the patient with carcinoma progress rather steadily. Second, the symptoms of a patient with cardiospasm are usually of longer duration than those of the patient with carcinoma. The roentgen studies with and without barium establish the diagnosis in the greatest number of patients. Occasionally the flat plate scout film may be confusing, suggesting a cardiac enlargement (Fig. 5A), a mediastinal lesion (Fig. 6A), or a pulsion diverticulum of the esophagus (Fig. 6B). However, these and other lesions such as esophageal peptic ulcer with fibrosis, congenitally short esophagus, hiatal hernia and epiphrenic diverticulum must be considered. They are usually self evident after fluoroscopic and esophagoscopic studies, although they may be present as associated lesions.

In a considerable proportion of cases of cardiospasm, the onset of symptoms occurs in the carcinoma age group and esophagoscopy thus becomes an essential diagnostic procedure. The experience of Garlock[15] deserves quotation, "I have seen smooth conical obstruction at or near the cardia which indicated probable benign cardiospasm. Yet the biopsy specimen obtained at esophagoscopy disclosed the presence of a neoplasm." In addition to confirming the diagnosis, esophagoscopy allows the mucosal changes to be assessed before treatment is instituted by the passage of the instrument.

The role of esophagoscopy in the diagnosis of cardiospasm has aroused much conflict of opinion. Olson[12] in his very complete and extensive survey of 601 cases of cardiospasm observed at the Mayo Clinic indicated that in his opinion esophagoscopy was not always an essential step in the diagnostic study of the patient whose x-ray showed the typical picture of cardiospasm. On the other hand, from observations made in the series herein presented, not only such lesions as carcinoma required differentiation, but it was felt that dilatation therapy should not be begun without first evaluating the degree of inflammation and possible ulceration at the cardia. Consequently, esophagoscopic studies are considered a routine and essential step in establishing the diagnosis of cardiospasm, as well as in controlling therapy.

ESOPHAGOSCOPIC PICTURE

Eosphagoscopy is frequently difficult in these individuals due to retained secretions and undigested food. At times extreme amounts of material must be aspirated before a satisfactory examination of the mucosa can be made. Amounts in excess of a liter are not uncommon and in these instances a large bore open-end aspirator made to pass easily into a 9 mm full-lumen esophagoscope is of great ad-

Fig. 4.—Tight fibrous stricture at the cardia with severe esophageal obstruction. Dilatation with a string-guided pneumatic bag gave prompt relief of symptoms.

vantage. Preliminary postural drainage assists in emptying the esophagus as well as the tracheobronchial tree prior to esophagoscopy. A lavage of the esophagus is of further help.

Mucosal changes are most often encountered in extreme degrees of megaesophagus with long standing retention. At esophagoscopy the mucosa is seen thrown into huge redundant folds and presents a granular, exudate covered surface with varying degrees of superficial ulceration due to the secondary esophagitis. Where elongation and tortuosity of the distal esophagus have occurred there may be difficulty in locating the cardiac orifice and in many instances acute angulation may prevent safe passage of the instruments (Figs. 2 and 3). When the cardia can be visualized in early cases, the passage of the esophagoscope into the stomach is met with little or no resistance, and the appearance of the mucosa may vary little from normal esoph-

agoscopic findings. Only the minimal dilatation and redundancy of the folds will be observed. This is in contrast to the organic fibrous stenosis of the late cases to which Mosher[3] referred. The zone of fibrosis may be only one centimeter in length, but in some cases may consist of 5 to 6 cm of tortuous, angulated esophagus requiring a string guide to permit even the simplest type of esophagoscopic inspection or dilatation (Fig. 4).

Foreign body episodes are by no means uncommon and are easily recognized. The patient usually volunteers the history of a hurried meal and can give the exact moment when a previously mild degree of dysphagia changed to complete obstruction with increased salivation. Eleven foreign body episodes occurred in nine patients in this series. In all but one instance this was a bolus of meat impacted in the cardia and requiring esophagoscopic removal. The one exception was a fragment of chicken bone.

THERAPY

The value of drug therapy in early cardiospasm is unquestioned. Symptomatic relief is obtained through the use of belladonna derivatives and nitroglycerin; the emptying of the esophagus due to the influence of these agents can be observed under the fluoroscope. However, they are of little or no value in patients with a far advanced lesion who have a complete loss of esophageal tone or a zone of fibrosis at the cardia completely replacing normal muscle. Supportive therapy consisting of a diet high in vitamins with water-soluable vitamin preparations used wherever possible is essential. Capsules do not pass through the cardia but dissolve in the esophageal contents, giving the patient an objectionable taste which soon forces him to discontinue their use. Oily vitamin substances float on top of the food and fluid column in the esophagus for long periods of time where they can be observed on esophagoscopy, particularly if the procedure is done in the upright position. Mild sedation is of benefit to some patients. Others are able to eat a dinner with less difficulty if it is preceded by a high-ball, without ice. Ice water and other ice cold liquids, and strong alcoholic beverages, on the other hand, cause severe spasm and increase the obstruction at the cardia. It is imperative that a thorough investigation of the remainder of the gastrointestinal tract and the gall bladder be made to eliminate any pathology that may be uncovered. The frequency with which cardiospasm is associated with upper abdominal disease is well known and often complete relief of the dysphagia follows proper management of the abdominal lesion. In this series, eight peptic ulcers were found associated with the cardiospasm: one esophageal, two gastric and five duodenal. There was definite
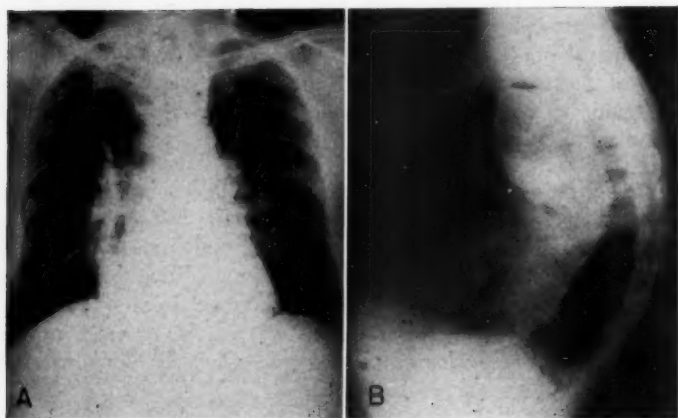
Fig. 6.—Extensive megaesophagus containing air. Fluid level is observed just above the diaphragm. B. Same patient (left lateral view) atfer barium administration. The upper part of the esophagus hangs over the arch cf the aorta to suggest a large pulsion diverticulum.
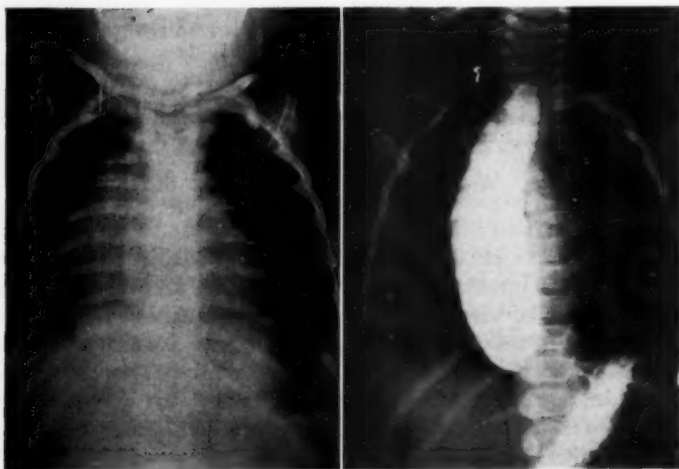
Fig. 5.—Cardiospasm in an infant. A. Posteroanterior chest film suggesting cardiac cnlargement. B. Same infant after administration of barium.

Fig. 7.—Pneumatic bag accurately placed in the zone of constriction under fluoroscopic guidance and inflated to dilate the stricture.

evidence of gall bladder disease in nine cases: three had cholelithiasis, three had had a cholecystectomy, in two the exact condition was not specified and in one there was a non-visualizing gall bladder.

Active therapy consists of intraesophageal dilatations and the external surgical procedures. The former may immediately follow the diagnostic inspection if acute inflammation and ulceration are not observed. In the presence of such ulceration it is felt that a delay of a week is of value, placing the patient on an ulcer regime of milk and cream, belladonna and bismuth powders. In early cases or those with a minimal amount of organic obstruction, dilatation by the passage of the esophagoscope into the stomach over a No. 10 bougie guide or a previously swallowed thread frequently gives dramatic relief. This may be followed by the passage of a Hurst mercury-filled bougie, No. 28 to 32 English, and later a Mosher pneumatic bag or a hydrostatic dilator. A combination Hurst mercury filled

bougie and pneumatic bag first described by Browne and McHardy[16] has also proven very successful.

The response obtained to such dilation procedures is usually very great. However, careful placement of the pneumatic bag in relation to the zone of obstruction, or absolute assurance that the mercury bougie actually enters the stomach, dilating the constriction are very significant points in technique. Thus, dilatation under fluoroscopic control (Fig. 7) is a procedure which leaves no doubt as to position, and improves results sufficiently to assure its value. The frequency with which the pneumatic bag or the Hurst bougie are seen to curl back on themselves in the esophagus while being observed under the fluoroscope is convincing evidence that often blind bouginage, even with a string guide, does not result in dilatation of the strictured zone itself. Best results are obtained if the pneumatic or hydrostatic bag is placed in the stricture over a string guide if necessary and the bag is inflated several times to the point of discomfort or epigastric distress indicated by the patient. To go beyond or to arbitrarily attempt to "iron out" the indentation in the walls of the bag by inflating it to a predetermined pressure measurement will result in an occasional esophageal leak or perforation. Actually, such a procedure is no more or no less than an intraesophageal muscle-splitting procedure. To dilate beyond the limits of elasticity of the mucosa will result in a mucosal tear. Two perforations have occurred in this series while undergoing pneumatic bag dilatation. Both patients survived, one following surgical repair and one responding to conservative therapy after the appearance of fluid in the left chest. In one instance the pneumatic bag became over-inflated in the stomach because of a leak in the inner rubber bag. It was deflated by passing an esophagoscope alongside of the stem of the bougie and puncturing the bag with a long Moersch esophageal varix needle.

The necessity for and value of surgical procedures for certain cases of cardiospasm that do not respond to intraesophageal procedures is unquestioned. Yet no single procedure can be considered suitable for all cases. The Heller muscle-splitting operation seems best suited for cases of tubular esophageal dilatation without evidence of a severe fibrous stricture if endoscopic measures have failed. More extensive procedures such as a long gastroesophageal anastomosis without resection of the cardia is required for the more extensive megaesophagus with markedly atonic walls. In this type of case fibrosis plays an important role. Finally, resection of the stricture and gastroesophageal anastomosis, which theoretically seems a logical procedure, results in the greatest number of failures due to regurgitation of gastric contents, esophagitis and stricture reforma-

TABLE III.

NO.	NAME	AGE	SEX	PREVIOUS ESOPH. OR DILATATION	TYPE OF SURGERY	NO. OF OPERATIONS	COMPLICATIONS
1.	MD	32	F	None	Heller	1	Esophagitis; stenosis
2.	ES	41	M	Repeated	Finney Cardioplasty Resection of stricture	2	Hemorrhage; stenosis
3.	CL	53	F	Repeated	2 Heller operations	2	Stenosis; good result after second operation
4.	RS	35	F	None	Resection of stricture	1	Stricture
5.	LR	60	M	Unknown	Three resections	3	Stricture recurrence after each operation
6.	TC	55	M	One	Unknown	1	Dysphagia; stenosis
7.	TG	40	F	None	Finney Cardioplasty	1	Stenosis
8.	CM	40	F	Repeated	Unknown	1	Stenosis
9.	MM	34	F	None	Heller	1	Stenosis; symptom free 3 yrs. postop. then return of pre-op. symptoms
10.	JC	25	F	None	Resection of stricture	1	Ulcer; hemorrhage; stenosis
11.	FW	29	F	Repeated	Esophagectomy	1	Postop. condition unknown
12.	BW	56	F	Repeated	Cardioplasty	1	Postop. stenosis
13.	JH	67	M	Repeated	Heller myotomy	1	Complete relief of symptoms
14.	EG	30	F	Repeated	Finney cardioplasty	1	Postop. condition unknown
15.	AK	53	M	Repeated	Heller	1	Stenosis
16.	BH	60	F	Repeated	Heller	1	Stenosis
17.	CM	19	F	Repeated	Unknown	1	Operative death
18.	FW	42	F	Repeated	Resection	1	Operative death

tion.[17] It is evident from a review of postsurgical recurrence of symptoms that in most cases, surgery directed toward alleviation of the obstruction is inadequate unless it includes a procedure designed to reduce the acid secretion of the stomach. This may be done by partial resection or by vagotomy. In the case of the latter procedure, a low gastroenterostomy is needed to assist in emptying the stomach.

That the problem of postsurgical recurrence and complications involving the esophagus postoperatively is a serious one is evident from a review of operated cases in the series of 189 patients herein reported. Admittedly, some patients, referred in the past for endoscopic dilatation and not seen subsequently, have probably been operated upon successfully for a recurrence of symptoms. However, of 18 patients who have had from one to three operations on the esophagogastric junction for cardiospasm, 13 have required postsurgical dilatation procedures identical with those preceding the operation, for recurrence of symptoms of dysphagia due to stenosis. There were two surgical deaths. Table III indicates the type of surgery performed, the number of operations and the complications. It is of interest to note that five of these patients had had no previous esophagoscopy or endoscopic dilatation and one had had only one esophagoscopic dilatation prior to surgery. The remaining 12 patients had had repeated pneumatic, Hurst or esophagoscopic dilatations without maintaining a satisfactory lumen through the cardia.

It becomes apparent when reviewing a series of patients with cardiospasm that the results of therapy, whether by endoscopic or external surgery, should not be judged for at least three to five years. Dramatic immediate relief is obtained by almost any type of active dilatation or surgical correction. However, recurrence rates following either type of management are high and cannot be judged by early, one-year response.

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REFERENCES

1. Mosher, Harris P.: On Being a Professor Emeritus, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 53:381 (Sept.) 1944.

2. Mosher, H. P.: Involvement of the Esophagus in Acute and Chronic Infection, Arch. Otol. and Laryngol. 18:563, 1933.

3. Mosher, H. P.: Infection as a Cause of Fibrosis of the Esophagus, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 50:633-662 (Sept.) 1941.

4. Kramer, P. and Ingelfinger, F. J.: Motility of Human Esophagus in Control Subjects and in Patients with Esophageal Disorders, Am. J. Med. 7:168-173 (Aug.) 1949.

5. Templeton, F. E.: Movements of Esophagus in Presence of Cardiospasm and Other Esophageal Diseases; Roentgenologic Study of Muscular Action, Gastroenterology 10:96-101 (Jan.) 1948.

6. Lendrum, F. E.: Anatomic Features of the Cardiac Orifice of the Stomach with Special Reference to Cardiospasm, Archives of Int. Med. 59:474-511 (Mar.) 1937.

7. Knight, G. C.: The Relation of the Extrinsic Nerves to the Functional Activity of the Esophagus, Brit. J. Surg. 22:155-168, 1935.

8. Knight, G. C.: Sympathectomy in the Treatment of Achalasia of the Cardia, Brit. J. Surg. 22:864-876, 1935.

9. Friedberg, Stanton A.: Observations on the Esophagus Following Vagotomy, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 59:751 (Sept.) 1950.

10. Jackson, Chevalier: The Diaphragmatic Pinch-Cock, The Laryngoscope (Jan.) 1922.

11. Mosher, H. P.: The Liver Tunnel and Cardiospasm, Laryngoscope 32:348-, 1922.

12. Olsen, A. M., Harrington, S. W., Moersch, H. J. and Andersen, H. A.: The Treatment of Cardiospasm: Analysis of a Twelve-Year Experience, J. of Thoracic Surgery p. 164 (Apr.) 1951.

13. Sabino, V. de F., Jr.: Megacolo E Megaesofago, No. Brasil Central. Resenha Clinico-Cientifica 19:3-15 (Nov.) 1950.

14. Benedict, Edward B.: Endoscopy, The Williams and Wilkins Co., Baltimore, 1951.

15. Garlock, J. H.: Progress in the Surgical Treatment of Carcinoma of the Esophagus and Upper Stomach. Cancer of the Esophagus and Gastric Cardia, C. V. Mosby Co., 1949, pp. 48-53.

16. Browne, D. and McHardy, G.: A New Instrument for Use in Esophago-spasm, J. A. M. A. 113:1963, 1939.

17. Holinger, P. H., Johnston, K. C., Potts, W. J. and DaCunha, F.: Conservative and Surgical Management of Benign Strictures of the Esophagus, J. of Thorac. Surg. 28:345-366 (Oct.) 1954.

XXVIII

THE VALUE OF THE LARYNGOFISSURE OPERATION

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Cancer of the larynx is but one aspect of a progressive disease which, if unchecked, always causes death. The major consideration in the management of the patient with cancer of the larynx is complete eradication of the growth in a determined effort to obtain a cure. Of secondary importance is preservation of vocal function. Both of these objectives can be achieved in selected cases of laryngeal cancer by an operative procedure known as the laryngofissure operation. Our experience with this procedure in 203 cases forms the basis of this report.

Desault,[1] who appears to have been the first to open the larynx by section of the cartilages, referred to the procedure as laryngotomy and recommended it for removal of impacted foreign bodies, laryngeal polyps and caries but did not suggest using it for laryngeal neoplasms. Laryngofissure was first used for removal of intrinsic carcinoma of the larynx in 1885 by Buck.[2] It was not until nine years later, however, when Butlin and Semon[3] reported favorable results, that the operation was accepted in the treatment of carcinoma of the larynx. Not only has it increased in popularity with time, but its great value in the treatment of intrinsic carcinoma of the larynx is fully appreciated and today it is accepted as one of the standard procedures for certain types of malignant lesions occurring on the vocal cords. When employed in indicated cases, few operations offer better results. Proper evaluation of the existing lesion on the vocal cords by means of mirror laryngoscopy or even direct laryngoscopy is therefore mandatory before attempts are made to remove the malignant lesion by this approach.

INDICATIONS

The indications for the laryngofissure operation as set forth by eminent laryngologists should be strictly followed. The ideal lesion

is a malignant tumor existing in the middle third of one vocal cord with evidence of normal appearing tissue on all sides. In such cases excellent results can be expected.

Frequently, however, a lesion may not conform to the ideal but may still be curable by the laryngofissure approach. For this reason, elasticity must be permitted in the selection of cases suitable for the laryngofissure technique. Thus, the life of the patient may depend on the wisdom of the laryngologist. It must be borne in mind that the first chance is the best chance of obtaining a permanent cure in patients with carcinoma of the larynx. The laryngologist is therefore beset with weighty decisions when a lesion is encountered on the vocal cords which is more extensive than the ideal case. Experience is the best teacher but even with this tremendous advantage errors will occur.

Lesions are often seen extending anteriorly on the cord, involving and crossing the anterior commissure and invading the opposite vocal cord. If involvement of the opposite cord is limited, removal of this extension plus a small amount of normal tissue, leaving the remaining uninvolved cord intact, will frequently give satisfactory results. This is true in spite of the fact that Broyles[4] showed that tendinous extension of the cords penetrated cartilaginous tissue at the anterior commissure. The only explanation possible is that the growth is superficial and does not involve the deeper layer of the cords.

Involvement of both vocal cords always taxes the judgment of the laryngologist, particularly if both cords are involved to the same degree. If a similar lesion existed on only one cord, there would be little or no hesitation in performing laryngofissure. With the lesion extending across the commissure and involving the opposite cord to practically the same extent, and with no evidence or suggestion of complications, the laryngologist is faced with a formidable problem. He should remember that a poor larynx is better than none. It is true that in such cases laryngectomy offers the best chance of permanent cure but at the sacrifice of the larynx. Would it be possible to preserve the larynx by performing bilateral laryngofissure with reasonable hope of completely eradicating the lesion? We have obtained surprisingly good results in such cases by removal of both vocal cords followed by extensive cauterization of the base. True, we have had recurrences but we have also had recurrences in patients with involvement of only one cord. It is extremely important to examine these patients periodically for several years postoperatively for possible recurrence. Removal of both cords simultaneously always provokes more reaction within the larynx and frequently this results in exces-

sive production of fibrous tissue, particularly in the region of the anterior commissure.

The laryngofissure operation may occasionally be employed for excision of benign laryngeal tumors which are too large for removal by direct laryngoscopy. We have also used this approach for extraction of impacted metallic foreign bodies and correction of persistent strictures of the larynx.

CONTRAINDICATIONS

Posterior extension of the malignant lesion with fixation of the cord and subglottic extension are always contraindications to laryngofissure. However, posterior extension to the arytenoid without fixation may necessitate, besides removal of the vocal cord, complete dissection and removal of the arytenoid cartilage followed by electrocoagulation. Good results in such cases have convinced us of the value of this procedure.

TECHNIQUE

The operative technique of laryngofissure which we have used over a period of years differs little from the standard technique employed by most laryngologists. Unless there is some justifiable contraindication, we prefer local anesthesia. An incision is made in the median line of the neck extending from the hyoid bone to just below the cricoid cartilage. By dissection the thyroid cartilage is exposed and then divided in the median line. Great care is exercised not to injure the perichondrium overlying the thyroid cartilage. Three or four drops of a 10 per cent solution of cocaine hydrochloride is injected into the larynx and trachea through the cricothyroid membrane. Invariably, this provokes the coughing reflex. To help control bleeding the cricothyroid membrane is then infiltrated with a one per cent solution of procaine containing six drops of epinephrine to the ounce. This is infiltrated along the projected line of incision.

The electric saw, though a great time saver, has been discarded because, in spite of all precautions, sequestration occurred too frequently, even months after the operation, as a result of the excessive heat generated, particularly when dense ossification of the thyroid cartilage was present. Much more time is consumed to divide the thyroid cartilage with the Clerf and Joseph saws but sequestration is avoided. As this line of division approaches the inferior border of the thyroid cartilage, it indicates exactly where the vertical incision should be made in the cricothyroid membrane. Insertion of a cotton applicator saturated with a ten per cent solution of cocaine through the opening in the cricothyroid membrane serves to cocainize the interior of the larynx.

The wings of the thyroid cartilage are then spread open to expose the interior of the larynx to direct view. The cordal lesion is examined and the limits of the tumor are carefully determined. We have found that an Alport retractor with a single tooth on each end provides an excellent view of the interior of the larynx. The affected vocal cord is separated subperichondrially from its attachment to the inner surface of the thyroid cartilage and a small strip of gauze moistened in cocaine is packed into the pocket created and remains there while attention is directed elsewhere.

Finally, with a slightly curved scissors the tissue above and below the cord is incised and a special pair of scissors with a 90 degree curve is used to separate the vocal cord in its posterior portion well beyond the visible border of the lesion. Vigorous bleeding is controlled with sutures, and the electrocautery is used freely to coagulate the wound. Meticulous attention is directed to suturing the line of incision in the upper portion of the larynx and to the cricothyroid membrane. Tracheotomy is never performed unless some complication develops.

COMPLICATIONS

Our records show three cases of postoperative hemorrhage necessitating reopening of the wound to control bleeding. Locating and suturing the offending vessel was sufficient in two cases, and the other patient proved to have a blood dyscrasia which necessitated not only reopening and packing the interior of the larynx tightly but also performing a tracheotomy and replacing lost blood.

The finding of small spicules of bone was not uncommon following use of the electric saw in spite of observing all precautions for cooling purposes. Since using the hand saw, we have not encountered this complication.

Secondary infection with a small fistula has prolonged healing in several cases. In an occasional patient extensive subcutaneous emphysema has developed as a result of severe coughing or vomiting efforts. In severe cases performance of tracheotomy has resulted in gradual subsidence of the emphysema.

All patients are warned that granulating tissue may develop within the larynx during the healing process. If it does not disappear in due course of time, it becomes necessary to remove the tissue either under indirect or direct laryngoscopy. If both cords have been removed, there is always considerable reaction resulting in development of excessive fibrous tissue. This tissue forms a synechia which occasionally narrows the laryngeal aperture considerably. On occasion it has been necessary to divide the synechia in the median

line and place a tantalum plate between the cut surfaces according to the McNaught technique.[5] This can best be accomplished by means of the suspension laryngoscope to expose the larynx to direct vision. The scar tissue within the larynx is incised in the median line with a laryngeal knife and excessive tissue is removed. The curved point of a No. 15 Bard Parker blade is placed in the most anterior part of the incision corresponding with the anterior commissure and the point is forced through the healed thyroid cartilage indicating accurately where the external incision should be made for the introduction of the tantalum plate. The incision in the thyroid cartilage should be just long enough to permit accurate introduction of the tantalum plate, which is then sutured in place. Removal of the tantalum plate after a few weeks usually results in a much larger laryngeal aperture with better breathing and improved vocal function.

RESULTS

Of 674 patients with carcinoma of the larynx encountered by us over a period of years, the laryngofissure operation was performed on 203. Recurrence developed in 31 or 15.2 per cent of these 203. Forty-four per cent of the patients with recurrences were salvaged by performance of laryngectomy as early as possible, and in a few cases block dissection of the neck was necessary. In 55 per cent of the patients with recurrences further surgical or radiation therapy was of no avail.

Fortunately, primary carcinoma of the vocal cords is usually of a low or intermediate grade of malignancy, a fact which accounts for the high incidence of cures obtained. Study of the gradation of malignancies in our series revealed impressive information which cannot be ignored. None of our patients with Grade 1 carcinoma of the larynx had recurrences. Recurrences developed in 13.4 per cent of those with grade 2 carcinoma, in 22.2 per cent of those with Grade 3 and in 75 per cent of those with Grade 4 carcinoma. Thus, the prognosis is much better for Grade 1 and Grade 2 lesions. It must be concluded from these figures that the surgical treatment of Grade 4 carcinoma of the larynx must be extremely radical.

Of additional interest is the study of results of treatment of lesions involving only one vocal cord and of those which have crossed the anterior commissure to involve some portion of the opposite vocal cord. Analysis of our cases showed that the opposite vocal cord was involved in 40 or 20.6 per cent of patients subjected to laryngofissure. The recurrence rate in this group was 26.9 per cent as contrasted with 12.2 per cent for those with involvement of only one cord.

Since the prognosis is obviously not as good when both cords are involved, the question immediately arises whether laryngectomy should be advised in such cases. Even when the lesion has spread beyond the anterior commissure to involve the anterior portion of the second vocal cord, our experience indicates that 73.1 per cent of the larynges can be saved. This salvage rate would seem to justify the laryngofissure operation in such cases.

A careful review of the cases of recurrences showed that in many instances the original lesion involved only a portion of one vocal cord and was therefore considered ideal for a laryngofissure operation. The same technique was used in all cases and we were confident that adequate tissue had been removed. The conscientious surgeon shares the chagrin and disappointment with the patient when a recurrence is manifest and instigates a study to determine, if possible, the cause of failure. If the growth within the larynx recurs in a short period following the operation, we are of the opinion that excision of the malignant tissue was inadequate. If, on the other hand, a growth appears in the larynx many years later, for example, 11 and 13 years after excision, as in two of our cases, we are inclined to consider these new tumors developing in the same organ rather than recurrences of the original lesion.

CONCLUSIONS

The great value of the laryngofissure operation lies in the fact that it affords an excellent approach to a malignant tumors which is relatively inaccessible. The ideal lesion for this approach is a cancerous tumor located in the middle third of one vocal cord with evidence of normal appearing tissue on all sides. A high percentage of cures can be expected in such cases if the lesion is completely excised.

Our experience indicates that the laryngofissure operation is indicated in approximately 30 per cent of patients with laryngeal carcinoma. Recurrences develop in only about 15 per cent of cases. The high incidence of cures can be attributed to the low grade of the malignant tumors in this organ.

We have been able to preserve comparatively good voices in 73 per cent of our patients in whom the lesion had spread across the anterior commissure to involve portions of the opposite vocal cord. In spite of removal of one vocal cord, excellent voices redevelop in many of these patients. A few have nearly normal vocal function. If the vocal process is disturbed when the cord is extirpated and the resulting fibrosis produces fixation of the cricoarytenoid articulation,

a weak voice can be expected. Also, failure of the operated side to "fill in" will result in a concavity which is responsible for a poor voice. In spite of removal of both cords, occasionally the resulting voice is surprisingly good. However, complete eradication of the cancerous tumor must take precedence over preservation of vocal function if this decision becomes necessary.

3503 PRYTANIA ST.

REFERENCES

1. Desault, Cited by Jackson, C. L.: Laryngofissure for Cancer of the Larynx, Arch. Otolaryng. 33:520-535 (Apr.) 1941.

2. Buck, Gurdon: Cited by Jackson, C. L.: Laryngofissure for Cancer of the Larynx, Arch. Otolaryng. 33:520-535 (Apr.) 1941.

3. Butlin and Semon: Cited by Jackson, C. L.: Laryngofissure for Cancer of the Larynx, Arch. Otolaryng. 33:520-535 (Apr.) 1941.

4. Broyles, E. N.: Anterior Commissure Tendon of the Larynx, Bull. Johns Hopkins Hosp. 70:90-91 (Jan.) 1942.

5. McNaught, R. C.: Surgical Correction of Anterior Web of Larynx, Laryngoscope 60:264-272 (Mar.) 1950.

XXIX

EXPERIMENTALLY INDUCED TEMPORARY THRESHOLD SHIFTS IN EARS WITH IMPAIRED HEARING

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The present paper is an introductory study of the measurement of threshold shifts by means of the Békésy audiometer[1] on subjects with hearing impairments associated with cochlear or neural pathology. Considerable data are available on changes in auditory sensitivity resulting from exposure of the normal ear to the various dimensions of wave form, sound pressure and duration of auditory stimuli. Relatively few comparable studies have been made on the pathological ear.

Gardner,[3] after a preliminary investigation tentatively concluded that "the fatigue of the conductively deafened observer was similar to the normal observer except the onset of fatigue was shifted by the amount of the threshold loss. For the nerve deafened observer, on the other hand, the onset of fatigue was found to occur at normal intensity levels. The occurrence of excessive fatigue in one of the nerve type impairment cases investigated appears to offer additional information on the nature of the lesion." At that time (1947) Gardner divided hearing impairment "into two main classes, namely, conductive or middle ear deafness, and nerve or inner ear deafness, with various combinations of these two types of impairment occurring rather frequently. Conductive deafness is caused by a mechanical malfunction of the ear drum or ossicular chain which interferes with normal transmission of the physical sound waves to the fluids of the inner ear; nerve deafness, on the other hand, is usually caused by atrophy of the nerve fibers which terminate along the basilar membrane or, to its physiological equivalent, degeneration of the hair cells of the organ of Corti."

Since Dix, Hallpike and Hood[2] pointed out that patients with hearing impairments resulting from labyrinthine hydrops experience

recruitment and those with impairments resulting from tumors of the eighth nerve do not show recruitment, it appears likely that atrophy of nerve fibres may not be physiologically equivalent to degeneration 'of the hair cells of the organ of Corti. Hood[4] conducted a limited study on "post-stimulatory fatigue in subjects with aural diseases" which also indicated a difference in fatigability between subjects with conductive and nerve deafness. Eight subjects participated in the tests. Two had conductive deafness, three had nerve deafness and three normal subjects served as controls. Each subject was exposed at 2048 cps for five minutes at an intensity level 100 db above the normal threshold value, and also at 4096 cps for five minutes 80 db above the normal threshold value. Hood found that "none of the subjects with conductive deafness or the normal controls showed any fatigue five minutes after the cessation of the fatiguing tone. In the case of the subjects with end-organ deafness, however, fatigue was present at both frequencies in all but one, a finding which supports the prevailing hypothesis that fatigue originates in the end-organs of hearing."

Hood also investigated "perstimulatory fatigue" in subjects with unilateral aural diseases, including 25 due to Ménière's disease and "a number of subjects with unilateral conductive deafness" by means of a simultaneous loudness level balancing technique. A tone of 2000 cps 70 db above its normal threshold value was applied to the normal ear. The intensity level at which this same frequency had the same loudness in the opposite pathological ear was determined by means of the alternate ear equal loudness level balancing procedure which is commonly used in the measurement of recruitment. "When a loudness balance had been established in this way, the tone in the impaired ear was maintained at a steady level and the loudness balance tracing taken from the normal ear in the usual manner. With cases of conductive deafness, the level of the balance tracing as would be expected was found to pursue a steady course . . . Quite different results were obtained, however, in the cases of Ménière's disease . . . with these nerve deaf subjects the balance tracing after a few seconds began to fall progressively to a new steady level. The rate and extent of the fall varied from subject to subject . . . We have interpreted this fall in the balancing level of the tone in the normal ear as clear evidence of an abnormal fatigability in the affected ear. Of the 25 Ménière's subjects tested in this way, 20 behaved in this fashion while with all tests carried out upon subjects with conductive deafness the balance tracing maintained a steady level . . . It is clear therefore that the existence of structural changes in the end organ seems to be associated with an increased susceptibility to perstimulatory fatigue.
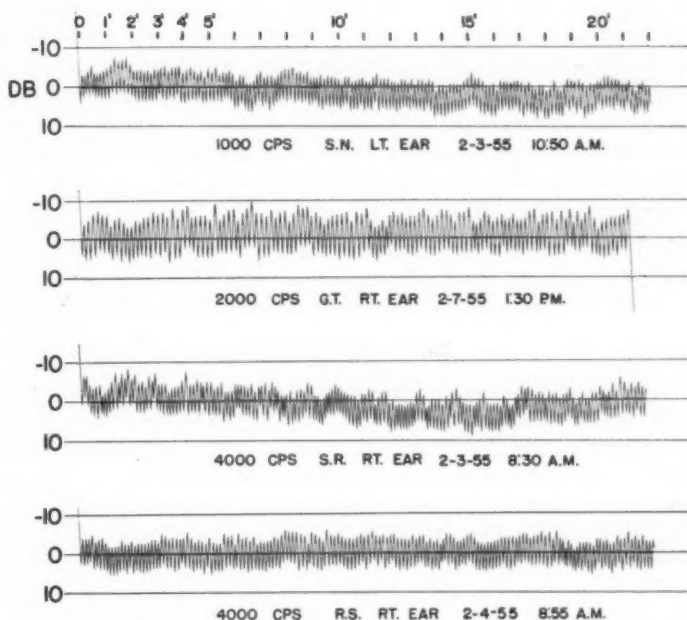
FIG. I. AUDITORY THRESHOLD SENSITIVITY MEASUREMENTS
ON TRAINED SUBJECTS

This we may regard as justifying the belief that the locus of perstimu-
latory fatigue is the end-organ itself."

Huizing[5] investigated the relationship between auditory fatigue
and recruitment. "To check this a comparison was made of auditory
fatigue in cases with recruitment and without. In order to attain
this the ear was stimulated over a period of three minutes at 2000
cycles/sec. and at an intensity level of 30 db above the *pathological*
threshold. The threshold for the same frequency was determined
again immediately after expiration of the stimulation period. These
experiments were made on a selected group of persons with respec-
tively: normal hearing, pure conduction deafness, pure inner ear deaf-
ness without recruitment and the same with recruitment, all observers
having about the same grade of hearing impairment (50 to 60 db loss).
The results may be classified as follows: threshold-shift in cases
with normal hearing 5 to 12 db and in cases of pure conduction deaf-

ness 7 to 12 db. In the cases of inner ear deafness without recruitment the shift was 5 to 11 db whereas the basin shaped threshold audiograms with recruitment showed a threshold shift between 19 and 22 db." Huizing also concluded that "the fact that the 30 db level above threshold in a case with recruitment corresponds to a much greater loudness value than without recruitment, explains that the magnitude of aural fatigue rather depends on the *loudness* value of the fatiguing tone than on its intensity."

The conclusions of the above three studies indicate that the pathological ear with so-called nerve deafness and especially the ear with recruitment is more susceptible to threshold shifts after exposure to pure tones than the normal ear or the ear with middle ear pathology.

INSTRUMENTATION

The Békésy audiometer[1] was used in the present study to ascertain the changes in intensity required to elicit a series of recurring thresholds responses over time intervals of up to 20 minutes duration and to obtain the pre-exposure and postexposure sensitivity to the pure tone stimuli. Each measurement was obtained at a single frequency level, with an attenuation rate of change of 2 db per second. The motor driven attenuator of the audiometer is of the continuously variable or stepless intensity type. The recording paper was driven with a speed of 8.3 mm per minute except for the measurements in Figure 3, in which the paper speed was 33 mm per minute.

A laboratory quality audiofrequency generator capable of delivering up to four watts of signal power to the audiometer attenuator was used as the sound source. In order to produce the relatively high intensity sound pressure levels required in the present study, a laboratory quality 20 watt amplifier was used between the output of the motor driven audiometer attenuator and the earphone. The earphone was of the moving coil type with an aluminum diaphragm, which was capable of producing appreciably greater undistorted sound pressure than moving coil earphones with plastic diaphragms. Three different experimental procedures were employed.

I. DECREASE IN THRESHOLD SENSITIVITY RESULTING FROM SUCCESSIVE THRESHOLD INTENSITY EXPOSURES

Figure 1 shows threshold sensitivity measurements on four different subjects over approximately 20 minute testing-time intervals. Each measurement was made at a single frequency level, indicated adjacent to the corresponding recording. The data shown in Figure 1 were obtained on subjects with normal hearing who had had considerable listening experience on the Békésy audiometer. It is ap-
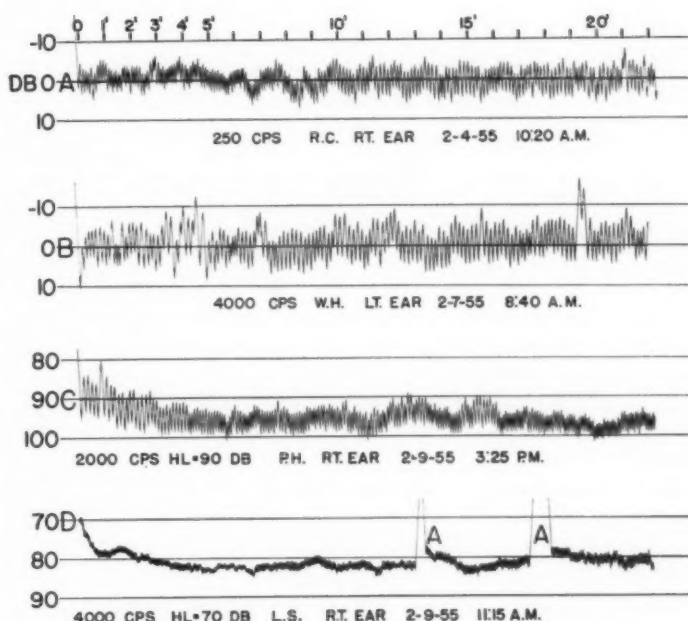
FIG. 2. AUDITORY THRESHOLD SENSITIVITY MEASUREMENTS
ON UNTRAINED SUBJECTS

parent that little or no shift in threshold sensitivity occurs for re-
peated threshold intensity exposures in the trained normal ear over
twenty minute testing time intervals as measured with the Békésy
instrument. As a matter of fact, a few comparable measurements have
been obtained on normal trained ears over a testing time interval of
one hour which show no appreciable threshold shift during the test-
ing interval.

Figure 2 shows comparable 20 minute threshold measurements
on four untrained subjects. A and B had normal hearing. C had
normal hearing up to and including 1000 cps and a loss of 90 db at
2000 cps of unknown etiology, the frequency at which the measure-
ment was obtained. D had a loss of 70 db at 4000 cps associated
with labyrinthine hydrops, the frequency at which this measurement
was made.

Measurements A and B of Figure 2 show little or no decrease in
threshold sensitivity over the 20 minute interval. The irregularities
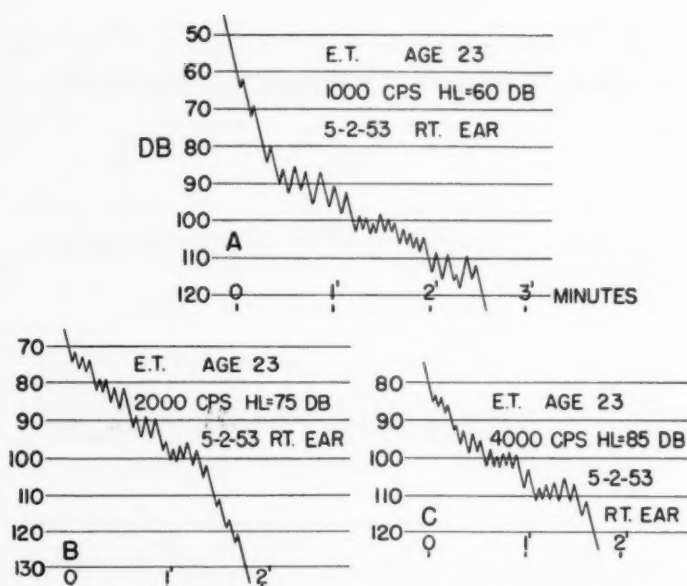
FIG. 3. AUDITORY THRESHOLD SENSITIVITY MEASUREMENTS
ON A PATIENT WITH AN EIGHTH NERVE TUMOR

of A and B (in comparison to the measurements on the normal ears in Figure 1) may be due in part to tinnitis, fluctuations in attention and changes in the subjects' criteria of hearing or not hearing the sound. C shows a relatively slow decrease in threshold sensitivity, most of which occurred during the first five minutes of the testing interval. D shows marked recruitment and a shift in threshold sensitivity of about 8 db at the end of the first minute of the testing interval, after which very little additional shift occurred. The two "A" breaks in D show the effect of brief interruptions of the 4000 cps tone to provide short rests for the ear under test. Measurable recovery followed the brief rests.

Figure 3 shows comparable measurements on a patient who had bilateral hearing impairments resulting from bilateral tumors of the VIII nerve. (This diagnosis was confirmed by subsequent surgery.) Note that in Figure 3 the recording paper was driven with a speed of 33 mm per minute instead of 8.3 mm per minute as in the other figures. No sound was perceived in this patient's left ear from a clinical audi-

ometer adjusted to reproduce its test frequencies at their maximum intensity output levels. The average loss in the right ear at 1000, 2000 and 4000 cps was 73.3 db. Measurement A of Figure 3 shows the threshold sensitivity measurement at 1000 cps, where the patient had a hearing loss of 60 db on the clinical audiometer. Inspection of the measurement shows a loss of threshold sensitivity of 50 db in approximately 2.5 minutes. B shows that at 2000 cps, where the patient had a loss of 75 db, a threshold shift of 50 db developed in about two minutes for threshold intensities. At 4000 cps, where the loss was 85 db, C shows that the patient developed a loss in threshold sensitivity of 30 db in about one minute and 45 seconds.

Such large and rapid decreases in threshold sensitivity appear to be characteristic of patients with hearing losses resulting from tumors of the VIII nerve. Reger and Kos[6] earlier reported this finding on a patient with a unilateral tumor, in which the opposite normal ear served as a control. Since then, three additional such patients have been tested in our laboratory, who, without exception, have demonstrated exceptionally rapid threshold shifts for threshold intensity levels. Threshold shifts of several decibels are observed in many patients who do not have losses resulting from tumors. However, the largest and most rapid shifts for threshold intensity observed in our laboratory have been those obtained on patients with hearing impairments resulting from VIII nerve tumors. Schubert,[8] using a somewhat different testing and recording technique, reported rather common occurrence of moderate threshold fatigue in ears with inner ear deafness. He did not report any measurements made on patients who had impairments due to VIII nerve tumors.

It seems foolhardy to speculate on the nature of the alteration in the hearing mechanism responsible for the threshold shift resulting from VIII nerve tumors until more pathological data are available. It may be assumed that many nerve fibers of the VIII nerve are nonfunctioning. Also that the pressure of the tumor may have interfered with the blood supply to the inner ear with a consequent reduction in metabolism and oxygenation of Corti's organ.

II. TEMPORARY THRESHOLD SHIFTS (TTS) FOLLOWING ONE MINUTE 20 DECIBEL SENSATION LEVEL (SL) EXPOSURES

This experiment repeats on pathological ears part of a study reported earlier by the authors[7] on the normal ear. In this study it was found that exposing the normal ear to a 1000 cps pure tone at a 20 db SL for one minute resulted in a TTS of 9.12 db for the 1000 cps exposure tone. This shift of 9.12 db is the mean value of 21

TABLE I.—MAGNITUDE OF THE TTS FOLLOWING ONE MINUTE
20 DB SL EXPOSURES AT 1000, 2000, AND 4000 CPS.

SUBJECT	1000 cps		2000 cps		4000 cps		RECRUIT-	RECOV-
	HL db	TTS db	HL db	TTS db	HL db	TTS db	MENT	ERY
1. D. D.	55	10.5	65	9.5	45	9.0	Marked	Normal
2. D. H.	60	10.0	55	8.5	60	9.0	Marked	Normal
3. M. D.	55	8.5	75	8.0	60	12.0	Moderate	Slow
4. G. M.	75	8.5	80	7.5	75	8.0	Moderate	Slow
5. B. K.	75	7.5	75	8.5	65	8.5	Moderate	Slow
6. N. P.	55	10.0	70	6.0	85	8.5	Slight	Slow
7. R. M.	65	8.0	65	12.0	65	11.0	None	Normal
8. M. E.	65	16.0	65	13.0	70	13.0	None	Slow
9. M. M.	80	6.0	70	7.0	75	7.0	None	Normal
10. P. N.	65	9.0	75	9.0	85	9.0	None	Normal
11. R. T.	70	9.0	70	9.5	70	10.5	None	Slow
Means	65.45	9.36	69.54	8.95	68.63	9.59		

measurements obtained on five trained and 16 untrained subjects.
(The mean of the shifts of the five trained subjects was 8.3 db.)

The data summarized in Table I were obtained on eleven selected
high school students of the Iowa School for the Deaf, Council Bluffs,
Iowa.

Only the students with cochlear or neural pathology who had air
conduction losses between 45 and 85 db at 1000, 2000 and 4000 cps
as measured with a clinical pure tone audiometer served as subjects.
Bone conduction threshold sensitivity measurements were obtained on
all subjects whose bone conduction losses did not exceed 60 db at 500,
1000, 2000 and 4000 cps, the maximum bone conduction loss that
could be measured at these frequency levels with the clinical audi-
ometer. Students whose hearing test, history or physical examination
indicated the presence of middle ear pathology were not used in the
study. The most frequent history of the students who served as
subjects was congenital deafness or deafness acquired early as the
probable result of an undiagnosed fever, whooping cough, measles,
mumps, smallpox or a "fall on the head." The influence of the Rh
factor was unknown.
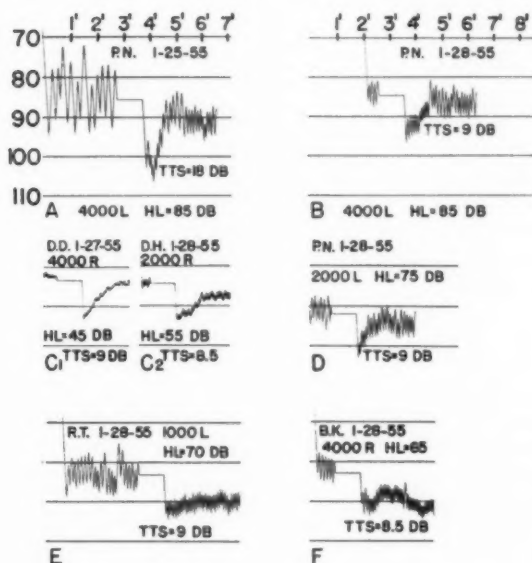
FIG. 4. TTSs FOLLOWING ONE MINUTE 20 DB
S L EXPOSURES

Each student was given one and sometimes two practice sessions on the Békésy audiometer on different days before the data shown in Table I were obtained. A and B of Figure 4 show the effect of practice by one of the subjects. The up and down excursions of the attenuator recording pen, to the left of the relatively short straight horizontal line, indicate the pre-exposure sensitivity. The horizontal line, 8.3 mm in length, indicates the duration of the one minute exposure. The exposure was initiated in the following manner: as soon as the subject had made a sufficient number of threshold responses to the tone with the signal key so that the threshold sensitivity appeared to be "stabilized," the experimenter pressed a timer control key when the recording pen reached a position midway between the peaks and valleys of the up and down excursions of the pen. This midposition indicates the mean value of the intensity range above and below the subject's threshold sensitivity. The timer was preadjusted to activate two different relays for a one minute period when its control key was pressed. The action of one relay stops the instantly

reversible motor which drives the audiometer attenuator and its asso-
ciated recording pen.   The second relay switches an external attenu-
ator out of the circuit.   This attenuator is connected between the
output of the audiometer and the earphone, and was adjusted to at-
tenuate the tone 20 db while the threshold measurements were ob-
tained.   The intensity level of the tone is increased 20 db above the
subject's threshold value while this external attenuator is switched
out of the circuit, thereby producing the 20 db SL exposure.   At
the end of the one minute exposure interval, the action of the timer
automatically and simultaneously starts the instantly reversible motor
and attenuates the tone to the pre-exposure intensity level.   The magni-
tude of the postexposure threshold shift is indicated by the difference in
intensity level between the pre- and postexposure threshold measure-
ments.

Two different practice effects are apparent in B of Figure 4.   The
up and down excursions of B are not as wide and the postexposure
TTS is about one-half as great as in A.   These two measurements on
the same subject emphasize the necessity of experimentation with
subjects only after they have had sufficient practice to permit reliable
observation or measurement.   C1 and C2 of Figure 4 show measure-
ments obtained on two different subjects who had marked recruit-
ment.   D shows a measurement in which the postexposure recovery
resembles that of the normal ear.   E shows a slow postexposure re-
covery and F shows no postexposure recovery over a three minute
interval.

After the last preliminary practice session each subject was given
a one minute 20 db SL exposure at 4000, 2000 and 1000 cps in the
order named.   (A 20 db SL exposure is an exposure 20 db above the
subject's threshold value, whatever that happened to be.   For ex-
ample, if a subject had a loss of 75 db at a given frequency level he
was given an exposure 20 db above this loss, which would be an in-
tensity level 95 db above the threshold value of the "normal" ear.)
Analysis of the data summarized and recorded in Table I shows
TTS's of the same order of magnitude as comparable data on the
normal ear.   The mean shift following the 20 db one minute ex-
posures at 1000 cps for the 11 pathological ears was 9.36 db.   The
comparable value for 21 normal ears was 9.12 db.   The shifts ob-
tained following the 20 db one minute exposures at 2000 and 4000
cps have about the same order of magnitude.   It therefore appears
that no significant difference exists between pathological and nor-
mal ears in the TTS following 20 db one minute SL exposures.   The
small size of the sample does not warrant detailed statistical analysis
or final conclusions.   However, certain relationships appear which are

of interest and which suggest future research problems. For example, the subjects with recruitment have shifts which closely approximate the mean value of the entire group. Subject No. 8 (M. E.) had the greatest shift for each of the three different exposure frequencies, indicating either a more tender ear or insufficient practice to eliminate variables responsible for shifts which are superimposed on the actual shift in the threshold sensitivity. There appears to be no systematic correlation between the magnitude of the hearing loss and the extent of the shift. The course of the postexposure recovery is worthy of note. The rate of the recovery was noticeably retarded in five of the eleven subjects and no recovery of threshold sensitivity occurred over a testing interval of several minutes in one subject.

III. THE EFFECT OF FIFTEEN MINUTE, SUB-THRESHOLD EXPOSURES ON SUBJECTS WITH SEVERE HEARING IMPAIRMENTS

Several high school students at the Iowa School for the Deaf were tested who responded to the lower frequencies generated by the audiometer only at relatively high intensity levels but who had losses exceeding 100 db for one, two or all of the frequencies at 1000, 2000 and 4000 cps. The effect on threshold sensitivity of exposing such ears to sound pressure levels a few db below the threshold value within this frequency range (1000-4000 cps) was investigated. For example, assume a subject with a 120 db hearing loss at 2000 cps. What would be the effect on his threshold sensitivity at 2000 cps of exposing him for 15 minutes to 2000 cps 10 db below his threshold sound pressure value? (This would be a pressure 110 db above the threshold of the normal ear.) Or what would be the effect on threshold sensitivity an octave higher than the subthreshold exposure frequency? Further assume that the subject did not have any middle ear or other type of ear pathology to interfere with the amplitude of the vibration of the fluids of the inner ear. Granted these assumptions, it is apparent that the 110 db sound pressure level above normal threshold at 2000 cps would vibrate the inner ear fluids with considerable amplitude. This sound pressure over an exposure interval of 15 minutes would produce an appreciable TTS in the normal ear for the exposure frequency, with probable greater shifts for frequencies from one-half to an octave higher than the exposure frequency.

Two different combinations of exposure and postexposure threshold measurements were obtained: (1) The "S" measurements consisted of a pre-exposure, subthreshold and postexposure series at the same frequency level. For example, A of Figure 5 shows an "S" type measurement at 4000 cps on a subject who had a 110 db loss at this frequency level. The left hand premeasurement shows the pre-exposure threshold sensitivity, the straight horizontal broken line
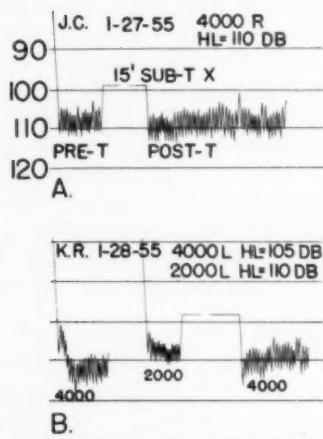
FIG. 5. EFFECT OF HIGH INTENSITY LEVEL SUB-
THRESHOLD EXPOSURES ON AUDITORY SENSITIVITY

shows the intensity level below the threshold value at which the
subject was exposed to the inaudible (to him) 4000 cps tone for 15
minutes and the postmeasurement shows the effect of this inaudible
subthreshold (but relatively high intensity level) exposure on the
threshold sensitivity. During this measure the subject was exposed
to a subthreshold intensity 10 db below his threshold level, which
would be an exposure 100 db above the normal threshold of audi-
bility at 4000 cps. As is apparent, this subject's threshold sensitivity
at 4000 cps was not altered by the 15 minute subthreshold exposure
at 4000 cps; (2) the "D" measurements investigated the effect of a
15 minute subthreshold exposure on the threshold sensitivity an octave
higher than the exposure frequency on subjects with severe impair-
ments for each of the two different frequencies. B of Figure 5 is a
typical "D" measurement which shows the effect on threshold sensi-
tivity at 4000 cps (where the subject had a 105 db loss) of a 15
minute subthreshold exposure 100 db above the normal threshold
value at 2000 cps (where the subject had a 110 db loss). The meas-
urement on the extreme left shows the pre-exposure sensitivity at
4000 cps, the adjacent measure shows the threshold sensitivity at
2000 cps, the straight broken horizontal line indicates the 15 minute
exposure level of the 2000 cps subthreshold tone in relation to the
threshold sensitivity at 2000 cps and the last measurement (on the

extreme right) shows the change in sensitivity for the 4000 cps tone following the 15 minute, 2000 cps subthreshold exposure.

The instrumentation for the "D" exposures was somewhat more complex than that of the "S" exposures. In the former, two different frequencies are involved. The relative positions of the 4000 and 2000 cps tones on the recording paper in B do not show the difference in threshold sensitivity between them. The gain setting of the audio-frequency oscillator, while producing the 2000 cps tone, was adjusted so that the subexposure level at 2000 cps would occur with the recording pen in a position ready to obtain the postexposure sensitivity at 4000 cps following the 2000 cps subthreshold exposure.

The results of ten "S" measurements (single frequency) and of ten "D" measurements (double frequency) indicate that the relatively high intensity level subthreshold exposures did not produce an appreciable change in the postexposure sensitivity, neither for the exposure frequency itself nor for frequencies an octave higher than the exposure frequency. However, two of the ten subjects showed rather rapid decreases in threshold sensitivity for threshold intensities following the subthreshold exposures. Several students in this group with severe hearing impairments experienced dizziness as well as sensations of discomfort and pain from the relatively high intensity sound pressures used while obtaining some of the threshold measurements. Interpretation of these observations is speculative without microscopic knowledge of the pathology responsible for the hearing impairments. It can be argued that the students who participated in this experiment could not have had normal cochleae, since the 15 minute relatively high intensity level exposures to which they were subjected would have produced definite shifts in the sensitivity of the normal ear. This viewpoint assumes that the temporary threshold shift resulting from this type and degree of exposure is an end organ phenomenon, which is generally thought to be the case. It is possible that this type of subthreshold relatively high intensity level exposure will facilitate differentiation between (a) the ear with a normal cochlea and a deficient VIII nerve, and (b) the ear with a deficient cochlea and a normal nerve. Of course it is possible that both the cochlea and the VIII nerve could be involved simultaneously. Unfortunately, the effect of subthreshold exposures was not tried on the few patients seen in the past who had hearing impairments resulting from VIII nerve tumors.

DISCUSSION

Experimentation with untrained subjects often yields incorrect results. Measurements must be repeated on a given subject until the

data show reasonably close agreement from measure to measure—reliability approaching or equaling that of the experienced observer. The experimenter also must never lose sight of the fact that the data obtained in a given situation are conditioned or influenced by the experimental technique employed in making the measurements. For example, recovery of threshold sensitivity following a sound exposure may and usually does occur more slowly when measured with a Békésy audiometer than when measured with a clinical audiometer, depending upon the technique used with the clinical instrument.

It is generally agreed that individuals with hearing impairments resulting from middle ear pathology respond in a manner quite similar to that of the normal ear following comparable sensation level exposures. The writers have seen no exception in the literature to the more or less general belief that individuals with hearing impairment resulting from nerve deafness experience greater temporary hearing losses than those with normal hearing or middle ear pathology following comparable sound exposures.

Certain pathological ears experience relatively rapid and marked threshold shifts from sound that can just barely be perceived—repeated or continuous threshold intensity exposures. This appears to be especially consistent in patients with hearing losses associated with VIII nerve tumors. However, decreases of a few db in threshold sensitivity to repeated or continuous threshold intensity stimuli are observed in many patients with other types of inner ear involvement. The degree or amount of the threshold shift within a certain time interval when measured on experienced subjects with an exactly reproducible method may be of value in differential diagnosis in the future.

The close agreement between the one minute 20 db SL exposures on the normal ear and the ear with other than middle ear pathology reported in this paper does not appear consistent with the results of other studies. The discrepancy may be due in part to the use of different exposure parameters and a different instrumental technique. Nor do the too few measurements reported herein support the thesis that subjects with recruitment experience greater threshold shifts than without recruitment. The one finding in this study which suggests a difference between the normal ear and the ear with inner ear pathology following sound exposure is the observation that recovery of threshold sensitivity following the exposure is slower in some of the pathological ears than in the normal ear.

It is apparent that much additional careful investigation must be performed on individuals with defective hearing resulting from defi-

nitely known pathology. Only then will the measurement of threshold shifts following sound exposures be of diagnostic significance.

UNIVERSITY HOSPITALS.

REFERENCES

1. Békésy, G. v.: A New Audiometer, Acta Otolaryng. 35:411-422, 1947.

2. Dix, M. R., Hallpike, C. S. and Hood, J. D.: Observations Upon the Loudness Recruitment Phenomenon with Especial Reference to the Differential Diagnosis of Disorders of the Internal Ear and VIIIth Nerve, J. Laryngol. and Otol. 62:671-686, 1948.

3. Gardner, M. B.: Short Duration Auditory Fatigue as a Method of Classifying Hearing Impairment, J. Acoust. Soc. Amer. 18:178-190, 1947.

4. Hood, J. D.: Studies in Auditory Fatigue and Adaptation, Acta Oto-Laryng. Supplementum 92, 1950.

5. Huizing, H. C.: The Relation Between Auditory Fatigue and Recruitment, Acta Oto-Laryng. Supplementum 78, 169-172, 1948.

6. Reger, S. N. and Kos, C. M.: Clinical Measurements and Implications of Recruitment, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 61:810-820, 1952.

7. Reger, S. N. and Lierle, D. M.: Changes in Auditory Acuity Produced by Low and Medium Intensity Level Exposures, Trans. Amer. Acad. Ophthalmol. and Otolaryngol. 58:433-438, 1954.

8. Schubert, K.: Horermüdung und Hördauer, Zeit. f. Hals-, Nasen- u. Ohrenheil. 51:19-74, 1944.

XXX

STUDIES ON THE OTIC LABYRINTH

VII. THE HELICOTREMA AND ITS RELATION TO THE DIMENSIONS OF THE BASILAR MEMBRANE AND PLACE THEORY OF HEARING

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The basilar membrane has, by actual measurements in histologic specimens,[3, 5, 7, 9] been found to narrow in its apical portion whereas throughout the rest of its length it widens progressively from the base toward the apex. A number of other structures—the tectorial membrane, organ of Corti, inner and outer rods, spiral ligament, spiral lamina—are all graduated in size. The place theories of hearing—specific tone representation in the cochlea and tone analysis in the cochlea—are based partly on the graduated size of the parts. The narrowing of the basilar membrane appears to be paradoxical, and, inconsistent with any place theory. Guild,[6] in a statement to the writer, gave the length of the narrowing portion as of the order of four millimeters, "and before reaching the narrowing portion, there is a section of a few millimeters which remains constant in width—a plateau, as it were." Wever[9] stated that most of the basilar membranes he measured, and particularly those of more than average length, attained a maximum width at a point about half a turn before the apical end, and then decreased rapidly (Fig. 1).

This anatomical peculiarity brings a number of questions to mind: Can such narrowing be consistent with place theories? Is it purposeful? Can it be related to a lack of firmness in the margin of the helicotrema as compared with the spiral lamina? Is the margin of the helicotrema actually more mobile than the spiral lamina? If so, is the margin of such a tiny aperture sufficiently long to cause, by its peculiarities, a change of dimensions in four, six or more millimeters of the length of the basilar membrane? Is the mere presence of a space-taking aperture pertinent? One would expect that it would have an effect upon the dimensions of the adjacent anatomic

parts and their dynamics.[4, 11] I do not recall having heard or read any discussion relating to this point. A search of the literature by title revealed nothing.*

Sutton and Schuknecht[8] have found experimentally that the results of needle trauma to the organ of Corti are different in the apical half turn than elsewhere in the cochlea. When a small needle point was introduced elsewhere into the scala tympani or against the spiral ligament and minimal pressure made, circumscribed islands of deafness resulted. If the same minimal pressure was used in the apical half turn, there was no demonstrable area of deafness. If the pressure was increased until deafness resulted, this was not a sharply circumscribed island. The effect was much broader and more diffuse. (Others,[1] it must be pointed out, have found that localized injury produced such widespread hearing loss that they have felt place theories are not tenable. The discrepancy in findings may be due in part to differences in methods of producing injury.)

In some way, as yet not fully determined, the width of the basilar membrane is related to frequency perception. It is not a matter of resonance as Helmholtz conceived it, but it is generally accepted, even in the modern compromise view of the place theory, that this increasing width is related to pitch. The tones in a harp or piano are evenly graduated in frequency, from the high to the low tones, by varying mass, length and tension. If, for structural reason, the strings had to be shortened in one zone, this shortening would have to be compensated by increasing mass or decreasing tension correspondingly. If a section of the frame to which the strings were attached were more mobile in one zone than elsewhere, that could be compensated for in a measure by shortening the adjacent strings to avoid an excessive drop in pitch. (There would, of course, be other marked effects resulting in damping and change of quality of tone.) If the graduation of frequency in the cochlea is to be constant and even for the full length of the basilar membrane and if this graduation depends in some way upon the width, mass and stiffness of the basilar membrane, then, if the attachment of one border is more yielding in a certain portion, as for instance at the helicotrema, it would be necessary, in order to keep the graduation even, to change the width, mass or tension in the area adjacent to this portion. Other effects from mobility in one attachment might not be pertinent because damping is already critical.

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*The Cumulative Index Medicus from Vol. 1 in 1927 through Vol. 52 in 1952 and the Armed Forces Current List of Medical Literature for 1953 and 1954 were searched and no titles of articles were found which dealt specifically with this subject.

Fig. 1.—Basilar membrane as measured and illustrated by Wever.[9] The widest point is reached at or before the beginning of the last half turn going toward the apex. Beyond this point the membrane narrows again. The dotted line has been added to indicate the region in which the hamulus and the margin of the helicotrema move with the basilar membrane.

If one looks at the lamina ossea in the last part of the middle turn and compares it with the margin of the helicotrema, it would seem that the latter must be more yielding. It *looks* as though the hamulus would be less firm than the rest of the lamina spiralis ossea, to which the margin of the basilar membrane is attached. It *looks* as though this lack of firmness would increase from the base of the hamulus to its tip. It further *looks* as though the lack of firmness would continue to increase beyond the tip of the hamulus through the membranous portion of the margin of the helicotrema.

This study was made upon formalin preserved, human cochleas through a window, cut into the apex, sufficiently large for purposes of observation of the helicotrema and somewhat more than the apical half turn of the cochlear partition. All measurements were made with a micrometer in the eyepiece of a Greenough type microscope. When calibrated against a standard blood chamber, it measured within 1½ per cent correct with a x 2 objective. The specimens

TABLE I.—MEASUREMENTS (IN MM) OF THE HELICOTREMA AND STRUCTURES IN THE APICAL HALF TURN OF THE COCHLEAR PARTITION. A, B, ETC. AS DESIGNATED IN FIGURE 2.

Specimen	A	B	C	D	E	F	G
139	0.95	0.65	0.65		0.45	2.15	
141	0.85	0.65	0.45		0.40	2.04	
144	0.90	0.60	0.30	0.55	0.50	2.35	1.17
148					0.60	2.30	1.20
152	0.95	0.75	0.60		0.60	2.22	1.10
154	0.85	0.60	0.40	0.65	0.80	2.20	1.20
155	0.75	0.55	0.40	0.55	0.50	1.95	0.95
156	0.80	0.45	0.40	0.75	0.50	1.95	1.05
157	0.95	0.75	0.55	0.70	0.50	2.25	0.95
158	1.00	0.80	0.60	0.70	0.45	2.275	1.20
160	1.00	0.60	0.40	0.45	0.30		
161	0.70	0.60	0.40	0.45			
165	1.00	0.70	0.55	0.45			
166	1.00	0.65	0.55	0.65	0.60	2.05	1.10
167	0.75	0.75	0.65	0.65			
170	0.80	0.70	0.50		0.55	2.325	1.10
AVG.	0.883	0.653	0.493	0.595	0.519	2.17	1.10

were mounted upon a specially designed clamp which contains a double universal joint permitting the placement of the specimen at any desired angle. (Clamp to be described in a subsequent publication.) A continuous flow of a film of fluid over the specimen kept the cochlea submerged at all times. In making measurements, the specimen was tipped in such a way as to bring the line of measurement as nearly as possible into a plane with the objective of the microscope. For some measurements it was found necessary to cover the specimen with a cover-slip to eliminate errors due to refraction of light in the covering film of fluid. Reasonable precautions were taken but it must be assumed that there is some error in the measurements because this factor could not be controlled entirely.

Taking Guild's statement—that the narrowed portion was about 4 mm in length—as a point of departure, the first step was to determine the length of the margin of the helicotrema to see if perchance it would be long enough to account for a change in width in such a length of basilar membrane. Others[2, 10] have made measurements of the helicotrema and given its diameter as about 0.5 mm and area 0.25 mm^2, but I found no mention of the length of the margin.

Fig. 2.—Photograph of the apical half turn of the cochlea, as seen through a large window. The superimposed letters indicate the measurements of the helicotrema, cecum and the organ of Corti which were used in making the calculations in the text. (A) the greatest length, i.e. from attachment of the margin of the helicotrema to the roof of the cochlea at the tip, to a point opposite this on the spiral; (B) the width at right angles to the measurement A; (C) the distance between the attachment of the lower end of the margin of the helicotrema to the modiolus and the point opposite on the spiral; (D) the height of the spiral, i.e. the distance from the lowest point of the margin to a plane through the point where the margin joins the roof at the apex; (E) the distance from the termination of the organ of Corti to the tip of the cochlear canal; (F) the distance from the outer margin of the tip of the organ of Corti across the helicotrema to the outer margin of the organ of Corti on the other side of the spiral—in other words, the width of the last half turn measured along the reference line X in Figure 3; (G) the radius of this half turn at right angles to the reference line.

TABLE II.—VALUES (IN MM) FOR THE LENGTH OF THE MARGIN OF
THE HELICOTREMA, LIMBUS AND ORGAN OF CORTI ARRIVED AT
BY CUTTING THESE STRUCTURES IN SITU RADICALLY FROM THE
MARGIN OF THE HELICOTREMA (SEE FIGURE 3) AND MEASURING
THE RESULTING SECTORS OF EACH STRUCTURE SEPARATELY,
THE SUM OF THESE SECTORS GIVING THE TOTAL LENGTH OF
THE STRUCTURE.

	LENGTH OF TIP OF STRUCTURE				LENGTH TO TIP OF CECUM			
SPECIMEN	HELI-COTREMA. MARGIN OF	LIP OF LIMBUS	TUNNEL	MIDLINE OF CORTI	HELI-COTREMA	LIMBUS	TUNNEL	MIDLINE OF CORTI
158		2.20	2.77	2.77	1.82	2.87	3.37	3.42
159		2.67	3.15	3.27	1.97	3.07	3.95	4.02
165		2.67	3.45	3.60	1.50	3.72	4.10	4.25
Averages	0.00	2.77	3.21	3.31	1.76	3.22	3.81	3.90

The dimensions of 15 (human) helicotremas were measured as
illustrated in Figure 2. The results appear in Table I. It is to be noted
that the course of the margin of the helicotrema is a rising spiral and
both the rate of rise and the turn of the spiral are inconstant—that
is, in following the course of the margin from the point where it
leaves the modiolus to its termination at the apex, both of these fac-
tors are found to vary. Therefore, accurate measurement and cal-
culation are difficult. Considering that the helicotrema is shaped
approximately like a somewhat flattened circle lying in an oblique
plane with a defect in the circumference equivalent to the measure-
ment D, calculating from the average in Table I, a value of 2.17 mm
is reached as the length of the margin of the helicotrema. This would
seem to be long enough to correspond with 4 mm of basilar membrane
but could not be considered as entirely accurate, therefore another
method was used.

Two models of the margin of the helicotrema were constructed
of soft wire using measurements A, B, C and D on a scale of 100:1.
After the wire was shaped, it was straightened out and measured.
One measured 168 mm and the other 170 mm. Taking the average
and dividing by 100, a value of 1.69 mm was found as the length of
the helicotrema, somewhat less than the 2.17 mm found by calcula-
tion. This result was probably more nearly accurate than the first
(2.17 mm) but still depended upon measurements of which one
cannot be entirely certain.

A different and simpler approach was devised: that of making
radial incisions in situ from the margin of the helicotrema through

Fig. 3.—Radiating lines along which the structures in the last half turn were cut with the scissors. X marks the reference line between the tip of the limbus and through the point where the margin of the helicotrema joins the modiolus. The sectors of the structures so produced were measured separately. The tips of the organ of Corti and the limbus were used as reference points rather than extending the incision through them. (H) hamulus; (L) limbus; (C) tip of the organ of Corti; (CE) tip of cecum.

the organ of Corti in the last half turn and measuring the sectors. Five or six such incisions were made in each of three specimens (Fig. 3). The point at which one can definitely say that the organ of Corti first becomes adjacent to the helicotrema is rather indefinite. An arbitrary spot had to be selcected. It was finally decided that a reference line, projected from the tip of the limbus through the point at which the lower margin of the helicotrema joined the modiolus, should determine the spot (X in Fig. 3). It was along this line that measurements F in Figure 2 were made. The number one incision was made along this line. These incisions were all made under a dissecting microscope with a special, very fine, eye scissors. The length of each of the sectors produced by these multiple incisions upon various structures was measured, i.e. the margin of the helicotrema, the lip of the limbus, the tunnel in the organ of Corti and the midpoint of the bulge of the organ of Corti. It was felt that these measurements were reasonably accurate. The results are given in Table 2.

A method of measuring very simply, and almost directly, the length of the midline of the basilar membrane suggested itself when it was noted that the reference line X in Figure 3 cut the organ of Corti into an almost perfect half circle. The outer border of the organ of Corti lies almost in the middle of the basilar membrane. The distance from the outer border at the tip of the organ of Corti across the helicotrema along the reference line X to the outer border on the other side, F Figure 2, is almost exactly twice the length of a line drawn at right angles to F from its midpoint to the outer border of the organ of Corti at the quarter turn. This latter line is then a radius of the circle of which F is the diameter. $\dfrac{\pi F}{2}$ would then give the length of the outer border of the organ of Corti and, almost exactly, that of the midline of the basilar membrane from the reference line to the tip of the former. The average value for F in 12 specimens was found to be 2.17 mm. $\dfrac{\pi\ 2.17\ mm}{2} = 3.408$ mm. The average value of E in Figure 2, the distance from the end of the organ of Corti to the tip of the cecum, in 13 specimens was found to be 0.519 mm (Table I). Adding E and F, a value of 3.93 mm is obtained for the length of the basilar membrane from the reference line to the tip of the cecum—a value which corresponds very well with the statements of Guild and the findings of Wever concerning the narrowing portion of the basilar membrane. This represents closely the distance in which the basilar membrane is adjacent to the helicotrema and is somewhat more than a half turn. It is noted, when observed through
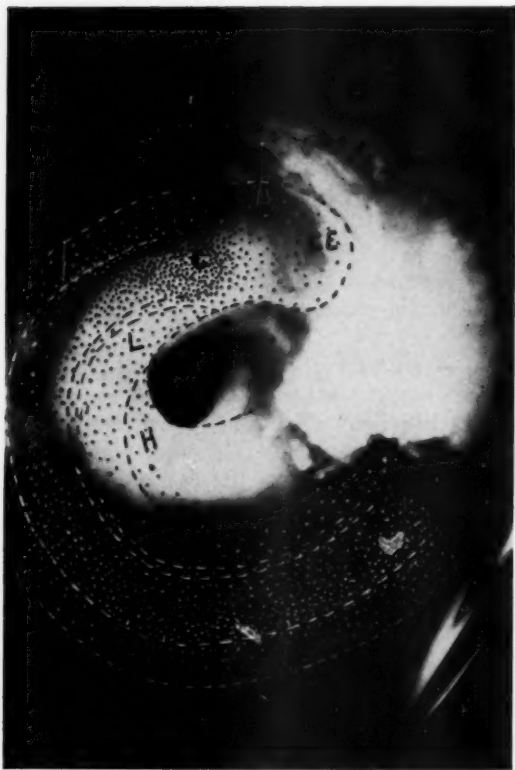
Fig. 4.—Structures in the apical half turn outlined in dotted lines on a photograph. Eleven human specimens were studied for motion and this illustration gives a composite picture of the result. When the helicotrema was corked and the cochlear partition made to swing up and down by varying the pressure in the scala tympani, the vibratory movements extended onto and included the hamulus, the tip of the limbus, and margin of the helicotrema. The stippling indicates the position of the motion and the number of dots indicates the amplitude—the thicker dots, the greater the amplitude. (H) hamulus; (L) limbus; (C) tip of the organ of Corti; (CE) tip of cecum.

TABLE III.—AVERAGE VALUES (IN MM) OF THE LENGTH OF VARIOUS STRUCTURES FROM THE REFERENCE LINE X (FIGURE 3) TO THE TERMINATION OF THE STRUCTURE AND TO THE TIP OF THE CECUM, AS DETERMINED BY VARIOUS METHODS AS INDICATED.

Structure	Length to Termination		Length to Tip of Cecum		
	By Measurement and Calculation	By Sectors	By Measurement and Calculation	By Wire Model	By Sectors
Margin of Helicotrema			2.17	1.69	1.76
Lip of Limbus		2.77			3.22
Tunnel		3.21			3.81
Midline of Corti		3.31			3.90
Outer Margin of Corti	3.408		3.93		

a microscope, that this portion of the basilar membrane and the margin of the helicotrema subtend the same angle.

OBSERVATIONS OF MOTION

Static measurements, whether in histologic sections or in situ, may be misleading. It is better to see the parts actually in motion. This can be done in the intact specimen (except for windows made for observation) by causing the cochlear partition to rise and fall by varying the pressure between scalae tympani and vestibuli by means other than sound (method to be described in a subsequent publication). There seems to be no essential difference in motion between fresh specimens and those which have been in formalin.

Eleven cochleas were opened at the apex in such a way as to make somewhat more than the final half turn of the cochlear partition visible. By making the partition move up and down, it was found that in this apical half turn, the width of the moving portion increases progressively almost all the way to the tip of the organ of Corti. This width of the moving portions does not decrease in a manner to correspond with the narrowing of the basilar membrane. This can only mean that other structures take part in the motion. It could be readily seen that this is the case. The limbus and part of the lamina spiralis were seen clearly to swing with the basilar membrane but to a lesser extent. The margin of the lamina spiralis begins to show motion about or somewhat before the point where the hamulus

begins. The width of that portion of the spiral lamina, i.e. the hamulus, which is involved in the movement, increases progressively toward the apex until the margin of the helicotrema itself is involved. From the tip of the hamulus onward, the membranous portion of the helicotrema continues to move up and down with increasing amplitude until the tip of the organ of Corti is reached. From that point on, the motion in the margin of the helicotrema and the basilar membrane decreases rapidly until it ceases entirely at the tip of the cecum. The up and down excursion of the organ of Corti increases progressively to its termination, at least in some specimens. The impression was gained that it would probably do so in all under normal conditions. Beyond the termination of the organ of Corti, as just stated, the motion in the cochlear partition decreases again (Fig. 4).

At the time these observations were made, a method for controlling the amplitude had not yet been developed. (Such a method has now been worked out and further results will appear in a subsequent publication.)   The excursions were, therefore, somewhat erratic both as to amplitude and width and measurements, consequently, somewhat uncertain.   Moreover, when the excursions on the inner side became progressively less until they were imperceptible, it was not always easy to decide upon an exact limit. Some attempts

TABLE IV.—MEASUREMENTS (IN MM) OF THE WIDTH OF THE
MOVING PORTION OF THE COCHLEAR PARTITION.

SPECIMEN	TIP OF CORTI	QUARTER TURN	HALF TURN	THREE-QUARTERS TURN
140	0.70	0.85	0.55	0.45
141	0.55	0.90	0.65	0.65
160	0.50	0.90	0.75	0.65
167	0.65	0.70	0.80	
Average	0.60	0.84	0.69	0.58

at measurements were made (Table IV). According to these, the greatest width of excursion occurred about a quarter turn back of the tip of the organ of Corti. This did not usually mark the greatest up and down movement. That seemed to be at or near the tip. The average widths of motion at the tip, a quarter turn, half turn and three-quarters turn back from the tip were respectively 0.6 mm, 0.84 mm, 0.69 mm and 0.58 mm. The maximum width of the fixed basilar membrane about a half turn back as found by Wever was 0.498 mm.

COMMENT AND SUMMARY

The problem of relating the narrowing of the basilar membrane in the apical half turn to the helicotrema began as rather complicated calculations of the length of the margin of the helicotrema. By using other approaches it became rather simple.

Summarizing the best measurements of the structures in the apical half turn from an arbitrary reference line drawn through the tip of the limbus and the deeper extremity of the margin of the helicotrema to the tip of the cecum, they were found to be: the margin of the helicotrema 1.76 mm, along the lip of the limbus 3.22 mm, along the tunnel 3.81 mm, along the midline of the organ of Corti 3.90 mm and along the outer margin of the organ of Corti 3.93 mm, the last figure by a different method than the other four. The last measurement would be very close to the length of that portion of the basilar membrane and is more than a half turn by the distance between the tip of the organ of Corti and the tip of the cecum. All measurements are consistent with the concept that the narrowing is related to the comparative mobility of the inner attachment of the basilar membrane in the region of the helicotrema. The narrowed portion subtends about the same angle as the margin of the helicotrema.

The observations of motion support the same concept and would even seem to account for the "plateau" where there is no change in width of the basilar membrane for a few millimeters since motion in the margin of the spiral lamina begins to appear somewhat back of the reference line X. The portion of the cochlear partition which took part in excursions did not narrow in the final half turn but continued to widen by including the hamulus and the margin of the helicotrema in motion. There was some narrowing near the tip of the organ of Corti but the amplitude—at least in some specimens—continued to increase by virtue of the membranous portion of the helicotrema, until the tip was reached.

The questions posed in the beginning can be answered largely in the affirmative. The narrowing can be consistent with place theories and may be purposeful. The margin of the helicotrema is more mobile than the spiral lamina and causes the inner margin of the basilar membrane in that area to be more mobile. The margin of the helicotrema is long enough to account for the narrowing since it subtends about the same angle, and also somewhat more, since the effect of the mobility extends somewhat into the middle turn. Sutton and Schuknecht's findings might possibly be explained on the basis of the more yielding character of the basilar membrane in the sector

adjacent to the helicotrema but there were undoubtedly other factors, probably more important, which influenced the results.

The chief results of this study can be quite simply given in a single statement: The width of the basilar membrane in the apical half turn does not delimit the dimensions of the portions of the cochlear partition which take part in motion.

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REFERENCES

1. Alexander, Irving E., Githler, F. J., Deiss, Andrew and Radwin, H. M.: Selective Cochlear Destruction, Cochlear Potentials, and Hearing Loss, A. M. A. Arch. Otolaryng. 61:46-53 (Jan.) 1955.

2. Bast, T. H. and Shover, Jayne: A Historical Survey of the Structure and Function of the Cochlea, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 52:279-329 (June) 1943.

3. Fernández, César: Dimensions of the Cochlear (Guinea Pig), J. Acoustical Soc. of Am. 24:519-523 (Sept.) 1952.

4. Fletcher, Harvey: On the Dynamics of the Cochlea, J. Acoustical Soc. of Am. 23:637-645 (Nov.) 1951.

5. Guild, S. R.: The Width of the Basilar Membrane, Science 65:67-69, 1927.

6. Guild, S. R.: Personal communication.

7. Hardy, M.: The Length of the Organ of Corti in Man, Am. J. Anat. 62:291-311, 1938.

8. Sutton, Samuel and Schuknecht, Harold F.: Regional Hearing Losses From Induced Cochlear Injuries in Experimental Animals, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 63:727 (Sept.) 1954.

9. Wever, E. G.: The Width of the Basilar Membrane in Man, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 47:37-47 (Mar.) 1938.

10. Wrightson, Sir Thomas: An Enquiry Into the Analytical Mechanism of the Internal Ear, with an Anatomical Appendix by Arthur Keith, London, MacMillan and Co., 1918, p. 249.

11. Zwislocki, Jozef: Theory of Cochlear Mechanics and Quantitative Analysis, Acta Oto-Laryngologica, Supplementum 62:1-76, 1948.

XXXI

THE TREATMENT OF ACUTE FRONTAL SINUSITIS

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The current treatment of sinusitis is based upon general principles of medical therapy utilizing the newer antibiotic, antihistaminic and related drugs, and the general principles of surgical drainage which are applicable to all of the sinuses regardless of anatomical position.

The frontal sinus, however, by virtue of its rather unusual embryological nature, and because of its relationships with critical anatomical areas, offers special problems and special challenges to the rhinologist.

The contributions of newer drugs and techniques have indeed modified some of the basic principles of the past. These modifications, however, have been both favorable and unfavorable; and it is necessary that the entire problem be re-evaluated at the present time in order to plan a rational therapeutic approach to this rather complex surgical anatomical problem.

1. PROPHYLAXIS

Prophylactic discussions are particularly appropriate in discussing frontal sinusitis inasmuch as diseases of this sinus are particularly based upon predisposition. The intricacies of the naso-frontal duct make predisposing factors of particular importance in this disease. Patients who are predisposed to recurrent attacks of acute frontal sinusitis should observe primarily two precautionary measure: (1) caution in diving and swimming in order to prevent the sudden ingress of water under high pressure into the frontal sinus through the naso-frontal duct, and (2) care in nose blowing during acute upper respiratory infection so as to avoid forcing of infectious secretions into the sinus. Adequate nasal shrinkage with an isotonic vasocon-

strictor should be used during upper respiratory infections to prevent blockading edema of the naso-frontal duct.

II. TREATMENT OF ACUTE SIMPLE FRONTAL SINUSITIS

Constitutional measures are of great importance in the basic treatment of the simple case of acute frontal sinusitis. These include adequate relief of pain through the administration of appropriate drugs. Antibiotic therapy should be instituted at once; penicillin is still our most valuable drug inasmuch as staphylococci are the most frequent causative organisms. Nevertheless, this drug should be avoided in patients with a definite history of penicillin sensitivity. In such instances, the utilization of either sulfonamides or members of the "mycin" family would be advisable.

Of great importance is the prompt usage of antibiotic drugs, and administration in sufficient doses to combat the disease process. Acute frontal sinusitis should be considered a medical emergency, and the patient should be put to bed and kept on adequate fluids, high protein diet, and in a room of controllable temperature and humidity.

Local treatment is of great importance in the early care of acute frontal sinusitis, and should be primarily directed toward adequate shrinkage of nasal mucosa to accomplish prompt ventilation and drainage of the frontal sinus through the naso-frontal duct.

Intensive nasal shrinkage should be accomplished by the use of a combination of a sympathicomimetic vasoconstrictor, along with the use of cocaine hydrochloride. A spray of 4% cocaine, with an equal part of 2% ephedrine is useful. Intensive nasal vasoconstrictions should be employed at least every two to three hours and should be followed by hot packs over the frontal and maxillary sinus areas.

In addition, the oral administration of propadrine HC1 25 mgm and pyribenzamine 25 mg every 3 to 4 hours is very helpful in maintaining shrinkage.

If there is obvious restriction of ventilation and drainage from the middle meatus by a laterally displaced middle turbinate, it should be infracted medially toward the septum under local anesthesia.

In some cases, it is frequently helpful to gently insert a properly curved cannula into the frontal sinus via the middle meatus for gentle irrigation of the sinus with normal saline or Ringer's solution. Such gentle irrigation may frequently allow for the expulsion of a large blockading accumulation of mucopus, following which there is frequently distinct subsidence of the infection with relief of pain and drop of fever. Such cannulation, however, should be done with the

gentlest of manipulation and should never involve forceful penetration of ethmoid cells.   Under no circumstances should a rasp be used.

In general, cannulation is a safe procedure in the average simple case of acute frontal sinusitis, but it is probably not advisable in the acute fulminating case, which will be described next.

III. TREATMENT OF THE FULMINATING CASE OF FRONTAL SINUSITIS

The patient with acute fulminating frontal sinusitis is usually one who has been swimming and in this situation an entirely different therapeutic attitude is required.   This patient should be considered dangerously sick until proven otherwise.   His treatment should be considered an emergency procedure, which should rank in seriousness at least with that of acute appendicitis.

The constitutional measures previously described under the simple acute type should be used as well in the fulminating case.   However, the dosages of antibiotic drugs should be sufficiently high so as to allow for immediate high concentration of the drug in the circulation. Even though surgical intervention may be obviously indicated, local vasoconstrictive measures should be instituted at once, eliminating the use of any instrumentation such as cannulation, which was described above.

The patient with the acute fulminating variety is best treated as a surgical emergency and the surgical procedure of choice is trephination of the frontal sinus by the external route.

Following completion of roentgen studies (designed primarily to determine the anatomic details of antero-posterior diameter, size, and location of the interfrontal septum) preparations for external frontal trephine surgery should be made.   The external technique is essential because it provides immediate and adequate external drainage from the affected frontal sinus through the compact bony floor, which is usually non-marrow containing and less likely to predispose to thrombophlebitis and osteomyelitis.

The dangers of complications in this disease are great.   Prompt external drainage is mandatory.

A.   Technique.   Although local anesthesia has been adequate in some patients, it is probably wiser to utilize general anesthesia with intratracheal intubation and intravenous sodium pentathol.

An incision is made below the inner portion of the eyebrow at the inner angle of the frontal bone.   This incision is made through

the skin, subcutaneous tissue and periosteum to the bone. The periosteum is carefully elevated over a very limited area so that no bone will be left uncovered by periosteum after the completion of the procedure.

An opening is then made through the floor of the frontal sinus disturbing only the thin compact bony floor. This opening may be made either with a small gouge or chisel or with a perforating burr, driven by a dental engine, or hand drill. The opening should not be made through the superciliary ridge or through the anterior wall since these areas are usually marrow-filled and may predispose to early osteomyelitis.

The opening is made large enough to admit a fairly rigid rubber or polyethylene tube of not less than 8 mm diameter. Usually pus under pressure will be encountered as soon as the sinus mucosa is opened. This pus may be gently aspirated and the tube inserted and sutured to the skin to maintain it in position. No manipulation is done in the region of the naso-frontal duct. A dry dressing is applied.

Such a trephine operation done early and combined with adequate antibiotic and supportive therapy will usually result in early subsidence of the acute infection, and prevention of complications.

The presence of meningeal signs in the absence of osteomyelitis is indicative of extension of infection through the posterior plate of the sinus. In such cases it is advisable at the same time as the original trephine to enlarge the opening adequately so that a portion of the posterior plate may also be removed in order to obtain adequate drainage of the extradural area, and to interrupt the process of retrograde thrombophlebitis.

The patient, following trephine surgery, should be carefully observed for the possibilities of extension to (a) the frontal sinus floor, (b) the anterior face, (c) the posterior plate, (d) the supra-sinus frontal bone, or (e) the orbit. The possibilities of retrograde thrombophlebitis extension to the meninges, the frontal lobe, or to any other portion of the cerebrum or cerebellum must be constantly considered.

The frequency of intracranial complication following the earliest invasive stage of frontal sinusitis calls for constant vigilance in these patients. The demonstration of intracranial disease may call for urgent neurosurgical intervention in the patient with acute fulminating frontal sinusitis. It cannot be emphasized too strongly that this patient is an acute medical and surgical emergency, and should be viewed with suspicion until all signs of the disease have subsided.

IV. THE TREATMENT OF CHRONIC FRONTAL SINUSITIS

Chronic frontal sinusitis is almost always the sequel of acute frontal sinusitis, and is usually due to some type of obstruction to the nasofrontal duct.

In chronic frontal sinusitis, pathological changes may be limited to the mucous membrane or may involve bone and periosteum. The extent of involvement will depend upon the virulence of organisms, the susceptibility of the mucosa to inflammatory change; but primarily on the permanence of nasofrontal blockade.

Allergy may play a part in chronicity and the acute inflammatory mucoiditis may produce by transition a change to hypertrophy and hyperplasia, and in some instances actual mucosal necrosis. In many instances of continuous empyema, there is a change to stratified non-ciliated epithelium with poor function. Submucosal fibrosis and ischemia may produce a non-functioning atrophic mucous membrane. In many instances, these mucosal changes may be accompanied by protective bony sclerosis and in some instances this protective action may be inadequate and result in sequestration. Sequestration may occur in the narrow region forming the typical findings of osteomyelitis and it may appear in the posterior wall on the floor in areas of osteitis.

The patient with chronic frontal sinusitis is primarily disturbed by a persistent dull headache which is usually quite troublesome.

The treatment of frontal sinusitis is largely surgical. Medical measures such as nasal shrinkage, irrigations and vaccines are only palliative and temporizing. Recently antibiotic therapy has yielded some encouraging results in the treatment of this disease and there are occasional reported cures without the necessity for surgical intervention.

Conservative therapy is limited to attempts at improvement in naso-frontal duct communication and attempts at antibiotic attack of the mucosal disease in the sinus proper. If such measures do not suffice, as they will not in most instances, some type of surgical intervention must be considered. In many instances several surgical procedures in rotation may be required before a therapeutic result is achieved. In the patient with chronic frontal sinusitis with moderate pain and no urgent distress, intranasal surgery should be considered wherever possible before external surgery is undertaken. The following intranasal procedures may be considered as conservative measures designed to increase ventilation and to lead to more adequate function of the naso-frontal communication.

A. *Correction of Anatomic Deformities.* These may include
(1) submucous resection of septal deflection and spurs, and (2) par-
tial middle turbinectomy to relieve middle meatus pressure by an
hypertrophied or cystic middle turbinate.

B. *Polypectomy.* Polyps of either allergic or infectious origin
should be removed if they obstruct the middle meatus.

C. *Anterior Ethmoidectomy.* The naso-frontal duct may open
directly into the nose or may be tortuous and drain into the cells of
the anterior ethmoid capsule. The removal of these cells will fre-
quently afford more adequate ventilation and drainage. Such cellular
removal may frequently make possible simple gentle cannulation of
the frontal sinus with irrigation and installation of antibiotic drugs.
The naso-frontal duct itself, however, should not be disturbed sur-
gically because of the possibility of subsequent fibrotic closure. In
this regard, it is important to remember the sage advise of the late
Dr. Harris P. Mosher, in which he cautioned rhinologists never to
disturb the virginity of the naso-frontal duct.

D. *Maxillary Sinus Surgery.* Inasmuch as the maxillary, an-
terior ethmoid and frontal sinuses all open into the middle meatus
concomitant infection in any of these sinuses may result in inflam-
matory changes leading to edema of the mucosa of the naso-frontal
duct. Consequently, treatment of the maxillary sinuses when indi-
cated is important in order to decrease inflammatory processes in
that area.

If none of the intranasal procedures, singly or in combination,
are sufficient to produce adequate drainage and relief of symptoms,
external surgery is necessary. External surgery should never be un-
dertaken lightly. The results in external frontal sinus surgery in
the past have never been too encouraging. Many patients who have
had external frontal sinus surgery are susceptible to complications
and the necessity for further surgical revision. However, when
symptoms persist and intranasal measures do not suffice, external sur-
gery is an absolute necessity in order to achieve drainage and relief
of retained secretions. The four primary indications for external
surgery are as follows:

A. *Relief of Continuous Pain.* Continued pain and headache
following conservative therapy and intranasal surgery is an indication
for the external surgery.

B. *External Frontal Fistula.* Fistulas of the frontal sinus usu-
ally form during the period of acute exacerbation of chronic frontal
sinusitis due to a blocked naso-frontal duct with a subsequent rupture

through the floor of the sinus. Such a fistula can only be closed suc-
cessfully by an external frontal operation.

C. *Mucocele or Pyocele.* Mucocele or pyocele of the frontal
sinus must be removed surgically externally in order to prevent its
recurrence.

D. *Intracranial Complications.* In all intracranial extensions
of frontal sinus disease, external surgery to the frontal sinus is man-
datory for therapeutic relief.

V. TECHNIQUES OF EXTERNAL FRONTAL SINUS SURGERY

Surgery of the external type to the frontal sinus is primarily
indicated for the removal of retained secretions, eradication of irre-
versibly infected sinus mucosa and measures to assure postoperative
adequate naso-frontal communications. The latter aspect of the
operation is undoubtedly the chief bottleneck and the chief cause
of failure in the history of external frontal sinus surgery.

A. *Conservative External Frontal Sinusotomy with Preserva-
tion of Naso-Frontal Duct.* This modification of the Lynch pro-
cedure has been found useful in our hands and consists of the follow-
ing steps:

1. Local or general anesthesia.

2. A Lynch incision in the region of the medial aspect of the
floor of the frontal sinus immediately below the eyebrow and over
the lateral wall of the nasal bone.

3. Elevation of the periosteum from the floor of the frontal
sinus and from the medial wall of the orbit. Care should be exercised
in this elevation in order to avoid damage to the lacrimal sac, the inner
canthal ligament and the attachment of the superior oblique muscle
pulley. As long as all of these structures are maintained in their
normal anatomic relationship as they are attached to the periosteum
there will be no change in their postoperative position and no dis-
turbance in function. The periosteum should not be buttonholed
during this elevation because of the danger of introducing infection
into the orbital tissues.

4. Bleeding from the anterior and posterior ethmoid vessels is
controlled either by ligation, pressure packing, or electrocoagulation.

5. The entire bony floor of the frontal sinus is removed fol-
lowed by complete removal of infected mucous membrane lining the
frontal sinus. Complete exenteration of the ethmoid cells, particu-
larly in the anterior and middle portions of the labyrinth is then
performed, working from both external and intranasal approaches.

The middle turbinate may be partially removed at the conclusion of the operation if it is enlarged or in any way obstructs the naso-frontal duct. *The naso-frontal duct itself, however, is undisturbed.* It may be gently inspected and probed but no attempt to destroy its lining mucosa should be made. In many instances this lining mucosa is not irreversibly damaged and upon removal of the pressure phenomena in the frontal sinus above and the institution of adequate external drainage below, such a mucosa may return to normal. No artificially created naso-frontal communication can serve the function of the frontal sinus as well as the undisturbed, patent virginal naso-frontal duct.

6. At the completion of the procedure, the cavity may be lightly dusted with penicillin powder. A Penrose drain is inserted and the wound is sutured, and a dry dressing applied.

In those instances where such conservative surgery is not adequate, modifications designed to create new naso-frontal communications must be considered. Should such a procedure as outlined above be insufficient for drainage, then the evidence is quite obvious that the naso-frontal duct mucosa is irreversibly diseased and will not return to normal. In such instances, measures to replace this communication must be instituted, although in our experience such measures are not frequently necessary.

B. *External Frontal Sinusotomy, with Reconstruction of Naso-Frontal Duct.* Although a number of methods have been suggested for the reconstruction of the naso-frontal duct, only a few of them have been proven to be of practical value. The introduction of inert materials has done a great deal to make successful reconstruction techniques possible. The utilization of a mucous membrane graft by Dr. Guy Boyden has been an important contribution to our armamentarium in the surgical attack in this disease.

Our experiences have primarily been in the realm of introduction of either tantalum of polyethlene tubes as artificial naso-frontal communications. Our present preference is for polyethylene because it is more easily manipulated to conform to the anatomic details and is virtually non-irritating (either chemically or physically) to the delicate structures in the region of the naso-frontal duct, and the middle meatus.

In utilizing polyethylene tube prosthesis, the previous conservative operation is carried out in the same detail as described, with the additional technique of removal of the naso-frontal duct mucosa which is undoubtedly irreversibly diseased and which has produced complete occlusion of the naso-frontal communication. The latter

is enlarged if necessary to allow adequate room for the insertion of a polyethylene tube of external and internal dimensions adequate to insure successful ventilation and drainage of the affected sinus. In general, an internal lumen of at least about 5 mm is desirable. A fairly thin wall should be used, but not one that will collapse as a result of external pressures. The tube should be so placed that it will adequately drain the lowest regions of the frontal sinus, and should not project up into the lumen of the sinus so high that adequate dependent drainage is impossible. Its intranasal position should not extend below the superior attachment of the inferior turbinate, and should not be in close contact with the nasal septum. Such contact may create a necrotizing effect upon the septum, with undesirable sequellae.

The operation is completed in the usual manner, as previously described, and adequate attention is paid postoperatively to hygenic measures intranasally to insure patency of the polyethylene tube. It is desirable to observe the intranasal region frequently to remove crusts and to maintain a patent tube at all times.

After a few weeks, the reaction in the region of the naso-frontal polyethylene tube subsides, and the tube usually remains as a fairly clean structure and allows adequate drainage and ventilation of the affected frontal sinus.

The employment of one of these procedures will usually suffice in the majority of instances to free the patient of the symptoms and in allowing a good cosmetic result. In rare instances, however, where one is confronted by huge sinuses with numerous pneumatic extensions, permanent and complete cure may be impossible unless an obliterative operation is performed on the frontal sinus. Such surgery is today rarely necessary. Where osteitis and persistent mucosal disease in the far reaching lateral recesses of such highly pneumatic sinus create persistent symptoms, an obliterative procedure such as originally recommended by the late Dr. Harris P. Mosher may be necessary.

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End of Special Mosher Memorial papers.

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XXXII

THE PATHOLOGY OF ACUTE DIFFUSE EXTERNAL OTITIS

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Acute inflammations of the skin of the external auditory canal occur frequently in hot, humid environments and have been variously named diffuse external otitis,[1] furunculosis, fungous ear, desquamative otitis externa,[2] tropical otitis externa,[3] and hot weather ear.[4] Although much has been written regarding the clinical picture and the bacteriologic flora associated with this disease, the pathogenesis of this entity remains a matter of controversy in medical literature.

As part of an integrated investigation of the etiology, pathology, and treatment of the external ear, a histopathologic study of the affected skin seemed desirable. To our knowledge biopsy material from acute cases of diffuse external otitis has in the past never been obtained, although an occasional report describing chronic changes in the periauricular area is available.[5]

On the basis of earlier and preliminary pathologic data, the following concept of the pathogenesis of acute diffuse external otitis was suggested by one of the authors.[6, 7] As a result of persistent high temperature and humidity, and wetting of the skin by swimming or bathing, there is produced in certain susceptible individuals a thickening of the stratum corneum. This results in the plugging of pilosebaceous units and the skin of the ear canal is deprived of its normal oily water-repellent protective coating. If the hot, humid period continues, there develops a pre-inflammatory state consisting of an itchy, dry, scaly skin, devoid of sufficient surface lipids. Such dry skin is constantly irritated by rubbing, scratching, and the application of irritating medications. Under such conditions the ubiquitous

TABLE I.—SUMMARY OF PERTINENT CLINICAL DATA REGARDING PATIENTS FROM WHOM BIOPSIES WERE TAKEN.

CASE NO.	SEVERITY	SEX	RACE	AGE	TIME OF ONSET	TRAUMA	SWIMMING	PAIN	DRAINAGE	Rx	DATE BIOPSY	PAST HISTORY	REMARKS
44	Severe	M	W	45	4 days	Syringed own ear	Yes	Yes	Yes	Al. acetate + alcohol	8/16/51	Recurrent every summer	------
129	Moderate	F	C	22	Months	------	------	No	Intermittent	------	11/14/50	Recurrent	Biopsy post canal wall. Pus encountered. Marked swelling lat. 1/2 canal.
127	Moderate	F	W	20	21 days	Cleaned ear with bobby pin	---	Yes	Yes	Aureomycin drops, topical	9/9/50	Neg.	Sagging of sup. wall and cobble stone effect
131	Severe	M	W	26	3 days	None	Yes	Yes	Yes	Al. acetate + alcohol	8/20/51	Recurrent "Fungus" Infections	------
126	Mild	F	W	27	4 days	Scratched ears	---	Yes	Yes	I. M. Penicillin + Streptomycin, oral Aureomycin	9/28/50	Chronic itching; digs at Ears; ear infection 1 yr. ago.	Cobble-stone effect Biopsy of sup. wall
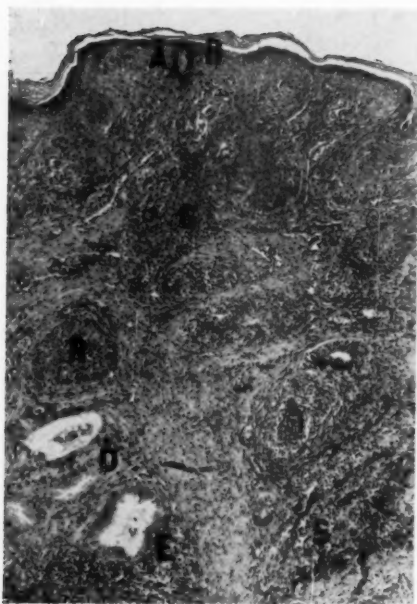
Fig. 1.—Low power view of epidermis and dermis showing destruction and degree of pathologic changes. A—Plug in follicle; B—Hyperkeratosis; C—Hyperplasia of Epidermal Cells; R—Epidermal prolongation in cross section; D—Apocrine gland with intraluminal exudate; S—Dermal infiltrate; E—Periglandular infiltrate.

Fig. 2.—Low power view of dermis and epidermis showing keratotic plugs and focal infitration. A—Superficial erosion and hemorrhage; B—Masses of cornified cells filling hair follicle; C—Hyperkeratosis; D—Hair shaft; E—Perivascular inltrate in dermal papilla and dermis; F—Sebaceous glands.

gram-negative bacilli are readily introduced. Since the bacteriostatic and fungistatic secretions from the epidermal glands are not present on the skin to retard the growth of these exogenous organisms, rapid superimposed inflammation occurs.

The present report is concerned with a more definitive study of skin biopsies obtained from cases which fulfilled the clinical criteria of acute diffuse external otitis. To serve as a control, a larger series of skin biopsies from normal ear canals, obtained at autopsies, served as a guide to the histology of this area.[8] Another group of skin biopsies, taken from the ear canal at the time of radical mastoidectomy, was studied and provided further comparison.[9]

MATERIALS AND METHODS

Biopsies were obtained from the external auditory canals of five patients presenting the clinical symptoms and signs of acute diffuse external otitis (Table 1). The ages of the patients ranged between 20 and 45. In two patients the disease was in the severe stage with complete obliteration of the lumen; in two cases the findings were classified as moderate; while in one there was partial control by local and systemic treatment at the time of biopsy.

The tissue was fixed in formaldehyde solution and mounted in paraffin. The specimens were cut in serial section (6 μ) and the majority stained with hematoxylin and eosin. Occasional sections were stained with the trichrome stain of Masson and by the Romanowsky technique.

Histopathologic detail was studied throughout the series of sections for each specimen, tracing the continuity of the processes through the depths of the tissue. The composition of inflammatory exudate was quantitated by differential counting. A search was made for micro-organisms in necrotic foci and in the lumina of inflamed glandular acini.

OBSERVATIONS

Epidermis. In all the sections examined striking changes were observed in the epidermis (Figs. 1-6). They were variable in severity and included hyperkeratosis, acanthosis, erosion, spongiosis, and parakeratosis. The total thickness of the epidermis was often greatly increased by cellular proliferation and edema. Hyperplasia of the stratum mucosum and the stratum germinativum often progressed to a striking degree in certain instances (Fig. 3). In the Malpighian layer the nuclei frequently seemed enlarged and vesicular with widely spaced and prominent chromatin granules. The cells were increasd

in number as well as size, and inter- and intra-cellular edema were prominent. The rete pegs were elongated and broadened, consisting of hyperplastic and hyperchromatic cells (Fig. 2). Mitoses were increased in number, and the dermo-epidermal margin was edematous and frequently ill-defined (Figs. 2 and 4).

The surface layer showed considerable thickening with irregular arranged masses of keratinized cells, many of which retained their nuclei (Figs. 3 and 4). These cornified cells sometimes filled the crypts and the mouths of the hair follicles (Fig. 2). Occasionally the superficial portion of the stratum corneum was lifted free, leaving small spaces which contained fluid (Fig. 3). In some areas there were small clear spaces constituting minute vesicles. Occasionally a micro-abscess was found within the epidermis (Fig. 4). Neutrophils and eosinophils could be found diffusely infiltrating all the layers of the epidermis (Figs. 3 and 4).

Foci of erosion were frequently observed (Figs. 4, 5, 6). In some instances, a necrotic layer of epidermis could be seen or the entire layer might be missing (Figs. 2, 5, 6). The denuded dermis showed fragmented fibrous tissue, dilated capillary blood vessels, and an exudate, largely of erythrocytes and fibrin, which lay upon the surface (Figs. 2, 6).

Dermis The degree and character of the inflammatory changes in the dermis were fairly constant in all of the specimens and consisted of edema, hyperemia, leukocytic infiltration, focal necrosis, and fibroblastic repair. The intensity of these inflammatory phenomena was greatest in and around the capillary loops in the superficial part of the dermis.

The vascular changes, most severe focally, consisted of dilatation, engorgement, necrosis, thrombosis, and rupture (Figs. 6-11). The endothelial cells were swollen and reduplicated so that the vascular lumina were reduced or actually occluded. The pericapillary fibrous tissue was edematous and infiltrated by mononuclear and polynuclear leukocytes (Fig. 9). Small foci of necrotic cells were sometimes found immediately adjacent to the involved occluded capillaries, but no bacteria could be detected intra- or extracellularly in any of the inflamed or necrotic foci. Vascular changes were also demonstrable in arterioles and capillaries situated in the deeper portions of the corium (Fig. 7). Sometimes thrombosis and inflammatory infiltrate were found in vessels closely adjacent to the acini of apocrine glands (Fig. 11). The contiguous fibrous tissue was infiltrated by eosinophils and neutrophilic leukocytes and small foci of necrosis (Fig. 8).

SOPHIAN-SENTURIA

TABLE II.
DIFFERENTIAL COUNT OF INFILTRATE (1900 CELLS).

Case No.	44	126	127	129	131	Average
Types of Leukocyte	%	%	%	%	%	%
Eosinophils	12	17	37	23	31	24
Neutrophils	29	28	16	25	36	27
Lymphocytes	30	33	22	28	15	25
Large Monocytes	18	20	19	19	12	17
Unidentified	11	2	6	5	6	6
No. cells counted	300	300	500	300	500	

TABLE III.
SUMMARY OF PERTINENT PATHOLOGIC FINDINGS IN EACH OF THE BIOPSIES.

Case No.	44	126	127	129	131
1. *Epidermis*:					
Hyperkeratosis	+++	++	+++	+++	++++
Parakeratosis	+	O	++	+	+++
Acanthosis	++	+	+	+	++
Intercellular edema	+	+	+	+	++++
Intracellular edema	+	+	++	+	++++
Vesicles	O	O	+	O	+++
Miliary abscesses	O	O	O	O	+
Lengthening of rete pegs	+++	+	++	++++	+++
Epithelial hyperplasia	+++	+	+	++	++
2. *Dermis*:					
Vascular thrombi	+	+	O	+	+
Vascular inflammation	++	+	++	+	++
Vascular necrosis	+	O	+	O	+
Perivascular infiltrate	+++	++	+++	+++	++++
Diffuse infiltrate	+	+	++	++	+
Abscess	O	O	+	+	++++
Edema	+	+	++	++	+
Dilated lymphatics	+	+	+	++	++
3. *Glands*:					
Apocrine degeneration	++	+	++	++	++
Apocrine compression	+	+	++	++	++
Apocrine inflammation	++	+	+++	++	+++
Sebaceous gland involvement	++	+	O	+	++

The distribution of the infiltrate in the dermis was largely focal, tending to be perivascular (Figs. 1 and 2). There was also a diffuse infiltrate which involved both the superficial and deep corium (Figs. 1 and 9). In the milder cases only small perivascular infiltrates were found, particularly in the superficial cutis. In more severe cases the foci were larger and accompanied by intense diffuse infiltration. In the severest cases the foci were massive and some of them consisted of aggregates of neutrophilic leukocytes with liquefaction necrosis (miliary abscesses). Surrounding them was a dense but diffuse infiltration of lymphocytes and monocytes. The composition of this exudate was notable chiefly for its high percentage of eosinophil leukocytes. The percentages of various types of leukocytes exclusive of the abscesses are listed in Table II.

Only limited observations can be reported in regard to nerve fibers and filaments because the major part of the tissue was superficial and contained only terminal protions of nerves. No changes which can be clearly characterized were observed regularly. Sometimes a nerve ending was involved in an inflammatory focus, in which case it was intact but edematous (Fig. 10). Similarly many nerve fibers seen in proximity to inflammatory foci showed hyperplasia of the perineural sheaths.

Glands. The apocrine glands were intimately involved in the inflammatory process (Figs. 10, 11, 12). The lumina were usually devoid of secretion. A notable feature was diminution of the glandular lumina, with crowding of the epithelial cells so that they formed staggered rows. Some nuclei were enlarged and hyperchromatic. For the most part the glomeruli were imbedded in the exudate, but their epithelia and myoepithelia seemed to be intact (Figs. 1 and 10). A few leukocytes were sometimes observed in the lumen and within the walls. In some sections where suppuration was far advanced, polynuclear leukocytes were found in masses in the gland space (Fig. 12).

The sebaceous glands were often involved in the superficial edema and exudate, particularly when there was dense inflammatory infiltration in the contiguous dermis (Fig. 6). In the glands most severely involved, polynuclear leukocytes infiltrated at the margins of the glands (Fig. 7). The holocrine cells, however, were normal in size and shape and their cellular integrity seemed not to be affected.

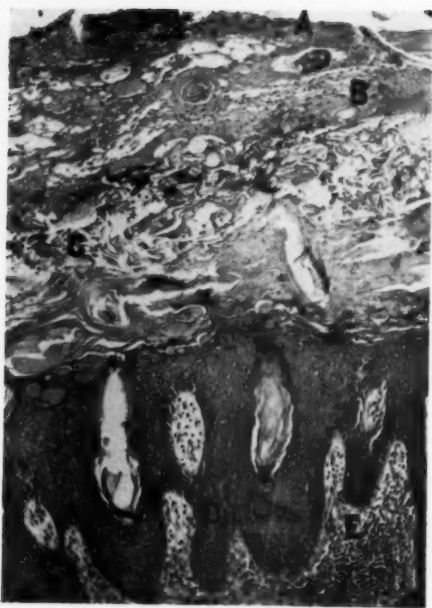The pertinent pathologic findings in each of the cases studied are listed in Table III.

Fig. 3.—Section through extremely altered epidermis. A—Parakeratosis;
B—Hyperkeratosis; C—Fluid accumulation in stratum corneum; D—Elong-
ated rete pegs; E—Dermal infiltration.

Fig. 4.—Section through epidermis showing micro-abscess.   A—Micro-abscess of epidermis; B—Parakeratosis; C—Dermal infiltration of leukocytes.
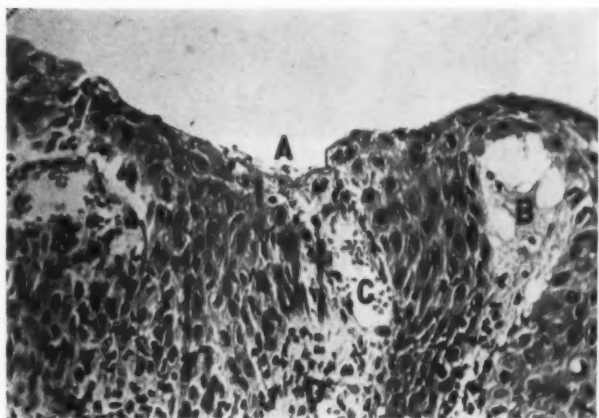

Fig. 5.—Section through epidermis showing erosion of surface (x 360).
A— Erosion of epidermis; B—Edema; C—Capillary close to free surface;
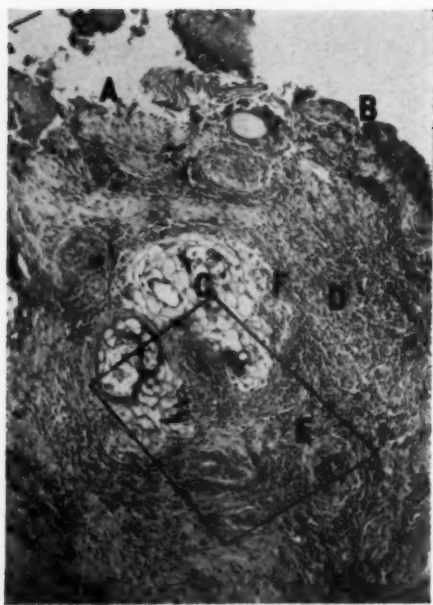D—Infiltration of leukocytes in dermal papilla.

Fig. 6.—Section through skin of ear canal showing ulceration of epidermis and inflammatory infiltration of dermis. E—Vessel showing endothelial proliferation and vasculitis; C—Sebaceous glands; F—Infiltration of margin of sebaceous gland; D—Dermal infiltrate; A—Ulcer; B—Organized exudate on surface.
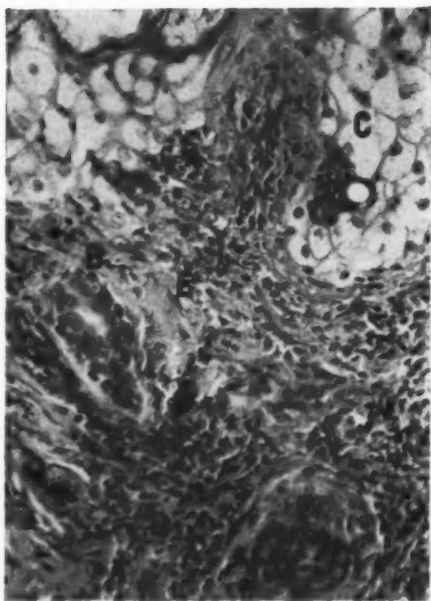
Fig. 7.—Enlargement of area outlined in Fig. 6 showing details of dermal infiltrate involving the capsule of sebaceous gland (200 x). E—Eosinophil leukocytes; C—Sebaceous glands; D—Inflammatory infiltrate; B—Duct of apocrine gland; A—Infiltrate in capsule of sebaceous gland; F—Vessel showing endothelial proliferation.

DISCUSSION

Within the limits of the restricted amount of pathologic material available for study, certain summarizations and evaluations may be made. Hyperkeratosis, edema, acanthosis, parakeratosis, and hyperplasia of the epidermis are uniform features of these skin biopsies. The epidermal lesions, which ranged from the mild to the very severe, demonstrated many features in common with atopic eczema and contact eczema.

The inflammatory process in the dermis has unusual characteristics. First, the capillary and arteriolar vessels showed dilatation, endothelial swelling, hyperplasia, and sometimes thrombosis and necrosis. These vascular reactions are of the type associated with toxic or allergic inflammations. On the other hand, they are rarely seen in purely pyogenic infections. Second, the exudate is notable for its high content of eosinophil leukocytes. This is at substantial variance with the type of infiltrate seen in common suppurative inflammations and from that previously described in changes of the skin of the external auditory canal associated with chronic otitis media and mastoiditis.

It is recognized that the eosinophil per se does not prove the allergic nature of an inflammatory process and that the clinical and morphologic picture of acute diffuse external otitis is not one usually seen in allergic disease. Nevertheless, there are certain clinical and pathologic aspects of the picture which are compatible with a state of sensitization. Among these are the recurrent nature of the process, the pruritus, the dry, exfoliative state of the skin prior to the acute exacerbation, the vascular changes, and the cellular composition of the exudate. For these reasons attention should be focused on the possible importance of altered tissue reactivity as a contributing factor in the etiology of acute diffuse external otitis.

The apocrine glands are often seen to be surrounded by the inflammatory process and sometimes infiltrated by leukocytes. In general the glands seem to be atrophic and decreased in size, and their epithelium gives the appearance of being non-functioning. The absence of dilatation or distention of the acini or ducts seems to exclude mechanical interference with drainage as a cause of the morphologic changes in the glands, although keratotic cellular masses often are found apparently filling the outlets of the pilo-sebaceous units.

It seems that compression of the apocrine glands may result from edema and infiltration of the periglandular fibrous tissue. It is also possible that interference with the vascular supply to the glands may be responsible for the degeneration of the epithelium

and its apparent lack of function. The sebaceous glands to a lesser degree are somewhat involved by the surrounding inflammation, but no conspicuous degenerative changes were observed.

SUMMARY

The histopathology of the skin of the external auditory canal of five patients with acute diffuse external otitis is described. The epidermis showed varying degrees of abnormal keratosis, hyperplasia, edema, and vesicle and abscess formation. The inflammatory process in the dermis had unusual characteristics, which included vascular inflammation, thrombosis, and perivascular exudation. The exudate was notable for its high content of eosinophil leukocytes. The apocrine glands seemed to be degenerative and non-functioning and secondarily involved by the surrounding inflammation.

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REFERENCES

1. Politzer, A.: Diseases of the Ear, Lea Brothers and Co., 4th ed., p. 173, Philadelphia and New York, 1903.

2. Daggett, W. I.: Desquamative Otitis Externa in Malta, J. of Laryng. and Otol. 57:427, 1942.

3. Beach, E. W. and Hamilton, L. L.: Tropical Otitis Externa, U. S. Nav. Med. Bull. 44:3:599-602 (Mar.) 1945.

4. Morley, G.: Otitis Externa: Hot-Weather Ear, an Investigation of 100 Cases and a Method of Treatment, Brit. Med. J. 1:373, 1938.

5. Williams, H. L., Montgomery, H. and Powell, W. N.: Dermatitis of the Ear, J. A. M. A. 113:641 (Aug. 19) 1939.

6. Senturia, Ben H.: Diffuse External Otitis: Its Pathology and Treatment, Tr. Amer. Acad. Ophth. & Otolaryng. 147-159 (Nov.-Dec.) 1950.

7. Senturia, Ben H.: Symposium on Medical Treatment of Otic Infections. Rationale of Therapy. To be published in Tr. of Amer. Acad. Ophth. and Otolaryng.

8. Sophian, L. H. and Senturia, B. H.: Anatomy and Histology of the External Ear in Relation to the Histogenesis of External Otitis, The Laryngoscope 64:772 (Sept.) 1954.

9. Sophian, L. H., Cooper, Z. K. and Senturia, B. H.: Pathologic Changes of the Skin of the External Auditory Canal in Chronic Otitis Media and Mastoiditis, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 63:261 (June) 1954.
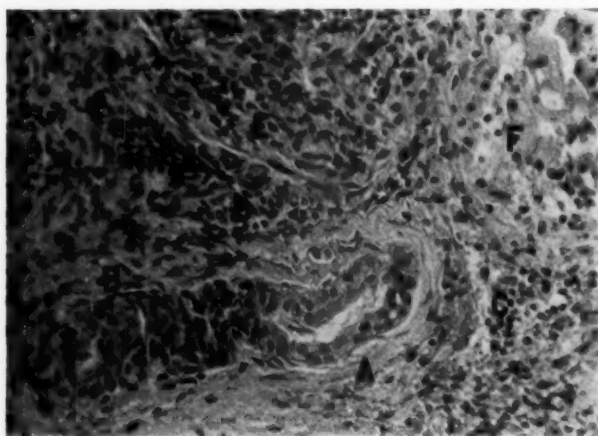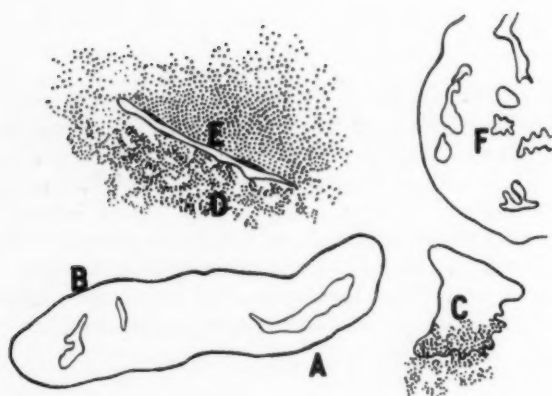
Fig. 8.—Section of skin of ear canal showing perivascular infiltrate and vascular necrosis in deep portion of dermis. A—Apocrine tubule; B—Apocrine duct; C—Ruptured necrotic vessel; D—Polynuclear leukocytes; E—Capillary; F—Degenerative apocrine gland.
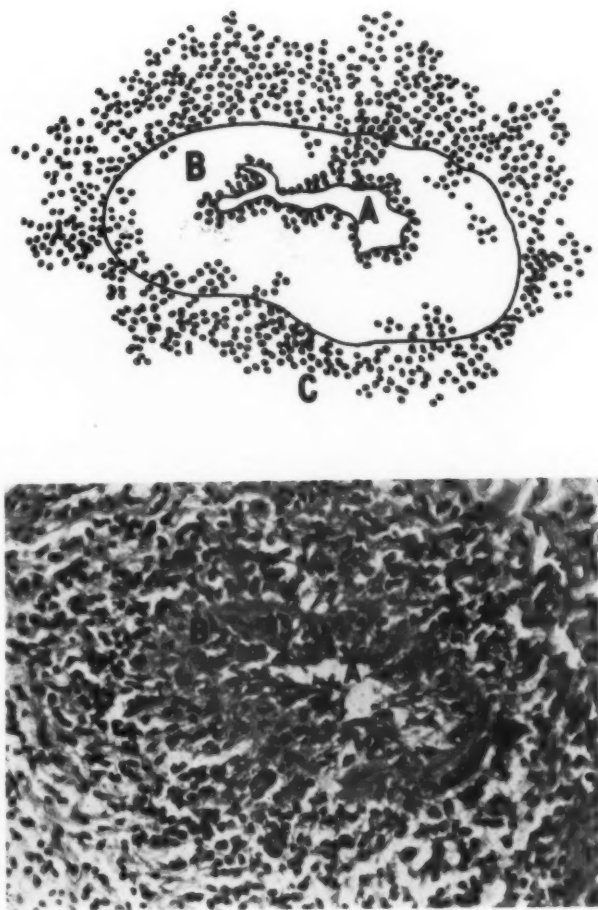
Fig. 9.—Section showing infiltration of vessel wall and perivascular cellular exudate in dermis (x 360). B—Polynuclear leukocytes within wall of vessel; A—Reduced lumen; C—Perivascular exudate.

Fig. 10.—Section through dermis showing diffuse periglandular and perivascular exudate. D—Inflammatory exudate; C—Arteriole of blood vessel, inflamed wall; B—Edematous nerve; A—Inflamed apocrine glands.

Fig. 11.—Section throught dermis showing thrombosed vessel adjacent to apocrine gland. B—Thrombotic and inflamed vessel; A—Apocrine gland; C—Dermal infiltrate; D—Focal infiltrate.

Fig. 12.—Section through dermis showing intra- and periglandular inflammation (x 150). A—Apocrine glands surrounded by inflammatory infiltrate; C—Infiltrate; B—Intraglandular exudate, polynuclear leukocytes predominate.

XXXIII

NEUROGENIC TUMORS OF THE LARYNX

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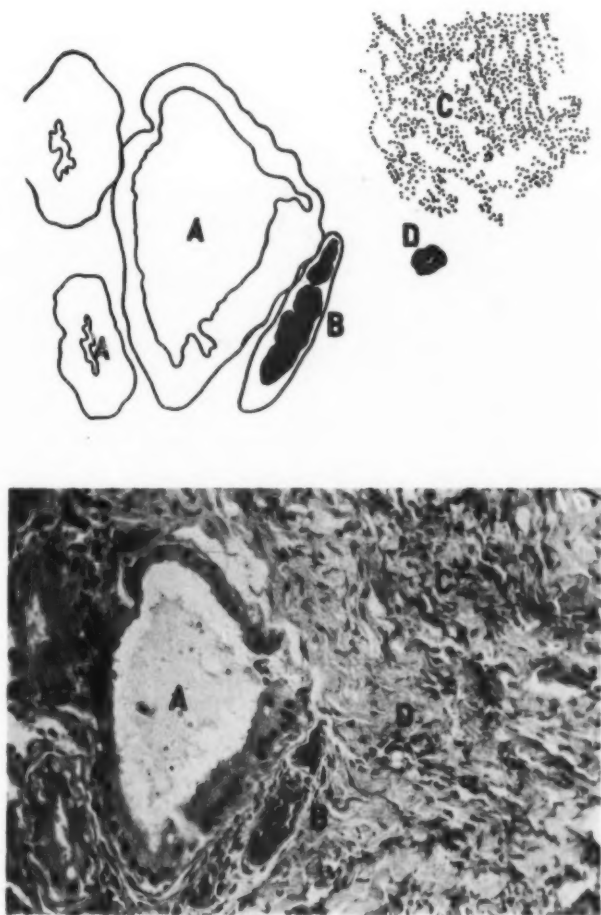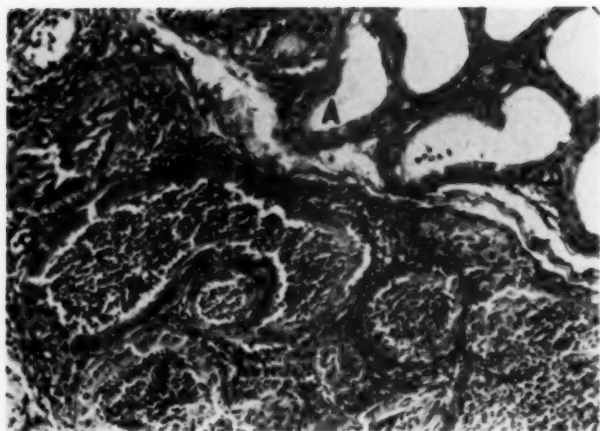Neurogenic tumors are decidedly uncommon in the larynx. The term is used to include reports both of neurofibromas and neurilemmomas, the neoplasms being intimately related. One case of ganglioneuroma under the title of "neurofibroma" has been reported by Eigler.[5] From the photomicrographs which accompany the report it is difficult to be certain as to the true nature of the tumor in question; no other reports of laryngeal ganglioneuromas were found. Similarly, amputation neuromas (not true neoplasms), although a distinct possibility, apparently have not been described in the larynx either. We have excluded from consideration certain tumors in which nerve structures form only part of the organic constituents, such as glomus tumors and hemangiopericytomas. We have not considered granular cell myoblastomas either, since the histogenesis of this neoplasm is still a matter of considerable dispute. The Schwann cells, which sheathe all the nerve fibers outside the central nervous system give rise to both neurilemmomas and neurofibromas. Considerable confusion has arisen from the multiplicity of terms used in the past to describe the same neoplasm. Thus, to describe the tumor known as neurilemmoma the terms Schwannoma, neurinoma and perineurial fibroblastoma have all been employed. The latter term was introduced by those who hold the origin of these Schwann cells to be from mesenchyme. Most authorities believe, however, that they arise from the neural crest and are, therefore, of ectodermal derivation (even though in tissue cultures they can produce connective tissue, a mesodermal derivative). The reasons given for considering neurilemmomas and neurofibromas as distinct entities are several. The neurilemmoma is usually a solitary tumor, but may be multiple and may even be associated with von Recklinghausen's disease (disseminated neuro-

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fibromatosis). It usually does not grow to large size, is always encapsulated and tends to become cystic when exceeding a size of three to four centimeters. It rarely becomes malignant (malignant Schwannnomas are said to be malignant from the start) and if adequately removed rarely recurs. Microscopically, there is an orderly arrangement of structure into: 1) A solid portion consisting of Schwann cells and delicate reticulin fibers arranged in cords, rows or true palisade fashion (palisading is by no means limited to nerve tumors, being frequently seen in smooth muscle neoplasms) and 2) a cystic portion in which spindle shaped nuclei are scattered in a disorganized manner among reticulin fibers. These two types of tissue arrangement in a neurilemmoma have been termed Antoni A and Antoni B respectively. The neurofibroma, on the other hand, is more frequently associated with generalized neurofibromatosis, lacks encapsulation, tends to grow to larger sizes and becomes malignant more often, especially when located in the deeper soft tissues.[25] Histologically, the Schwann cells proliferate in a disorganized manner instead of the usual orderly way seen in a neurilemmoma; Schwann cells and reticulin fibers are separated by abundant edema. Although these criteria appear clearcut, overlapping in gross and microscopic morbid features frequently presents considerable difficulty in assigning a particular neoplasm to either category. The presence of generalized disease apparently is no assurance that all the neurogenic tumors to be encountered will be neurofibromas. The common origin of both neoplasms from Schwann cells makes the distinction even more acute. This becomes apparent as one peruses the literature in search of reported cases and encounters discrepancies (according to the criteria enumerated) between the final diagnosis and the pathologic description of the tumor.

The available pertinent clinical and pathological data on 39 cases of laryngeal fibroma and neurolemomma have been studied. Two other cases briefly mentioned in a well known text[14] have been excluded from the review for want of clinical data.

Many reports of laryngeal fibromas are probably instances of neurofibromas,[21] so that the actual incidence of the latter probably well exceeds the usual estimates. New and Erich[19] reported only one neurofibroma among 722 benign tumors of the larynx. At the Jefferson Medical College Hospital five such neoplasms have been encountered in the period extending from 1929 to 1954. The other four cases have been reported elsewhere.[13, 24] Of the 39 cases, 24 occurred in females, 12 in males and in 3 instances the sex was not specified. The youngest patient was 5 years and the eldest 75 years of age. In six patients the laryngeal lesion was a manifestation of

Fig. 1.—Posterior view of the larynx showing the partly encapsulated neoplasm arising from the area of the left cricoid cartilage and pushing the epiglottis to the right. In the upper one third of the tumor can be seen an area of cystic degeneration and hemorrhage.

von Recklinghausen's disease.[4, 6, 9, 10, 12] Hoarseness has been the commonest symptom, varying in duration from a few months to as long as 20 years. The most frequent sites of the tumor have been the aryepiglottic folds, ventricular bands and true vocal cords. The largest previous tumor was that described by Walker;[29] it measured 4.5 x 3 x 3 cm and weighed 21 grams. In one case the tumor was histologically malignant.[21] The great majority of these masses have been easily shelled out endoscopically, the most radical procedure ever required being laryngo-fissure, with manual enucleation of the growth. In general, results of therapy have been satisfactory, recurrences being unusual.[21] One death from the tumor proper has been reported,[12] but clinical details were lacking. Residual vocal cord paralysis has been an uncommon sequela.[4, 9, 11, 29, 24] It may be pertinent to point out that vocal cord paralysis and obstruction of the airway have been recorded as manifestations of neurilemmomas arising in the pharynx as well.[23] The case of laryngeal neurilemmoma to be described represents the largest such tumor ever reported in the larynx and the only case in which removal of the growth has necessitated a laryngectomy.

REPORT OF A CASE

E. S., a 39 year old, married, white female was seen for the first time at Jefferson Medical College Hospital on September 10, 1953, with the presenting complaint of hoarseness of four months duration. The onset was rather sudden and unassociated with respiratory tract infection. She also experienced slight dyspnea on exertion and complained of making loud snoring noises during sleep. Physical examination upon admission revealed a markedly thin woman in some physical and mental distress. The heart and lungs were normal. The liver was externally palpable about two cm below the right costal border; there was an old, well-healed operative scar in the right lower abdominal quadrant. The spleen was not enlarged. Mirror laryngoscopy revealed a large, rounded, smooth swelling occupying the left side of the larynx and obliterating in part the glottic space, encroaching at the same time on the right side. There was no gross evidence of inflammation. The thyroid cartilage was believed to be broader than normal and externally had a nodular consistency, suggesting its invasion and erosion by the new growth. The trachea appeared displaced to the right. The hemogram and urinalysis were negative. A chest film revealed apical pleural thickening and displacement of the trachea to the right. X-ray films of the neck showed a large, cystic mass blocking the hypopharynx and pushing the epiglottis forwards, thus distorting considerably the normal anatomical arrangement of the laryngeal cartilages. Because of the dyspnea, a tracheotomy was performed shortly after the patient's admission. It was done under local anesthesia. The left side of the larynx was quite firm, suggesting a cartilaginous or bony tumor. It encroached upon the airway, almost completely occluding it. Ulceration was absent. Attempt was made to obtain material for biopsy, but it was largely unsuccessful due to the firmness of the mass.

On the eighth hospital day, and after explaining the situation to the patient and family, a laryngectomy under local anesthesia was performed by Dr. Louis Clerf. Considerable distortion of anatomical relations was found, the mass within the larynx extending up behind the body of the hyoid bone and also laterally to the left side of the neck. The thyroid gland was unusually vascular and friable and there was tracheomalacia of the upper two cartilaginous rings. The larynx was removed from above downward with a segment of the hyoid bone, the line of amputation being made between the third and fourth tracheal rings. The trachea was markedly softened and its wall swollen, producing considerable stenosis. The wound was closed and a drain inserted. The lesion in question was seen to arise probably from the left arytenoid or cricoid cartilage. The post-
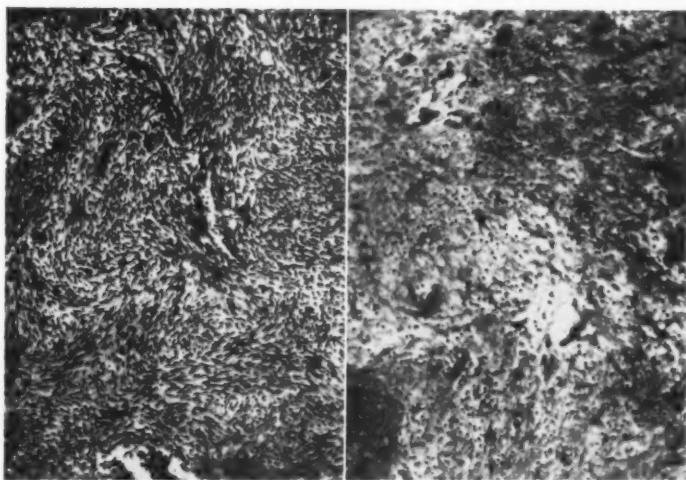
Fig. 2.—To the left, closely packed bundles of cells with spindle shaped nuclei (Schwann cells) and reticulin fibers (Antoni A type of arrangement). To the right, loose network of Schwann cells and delicate connective tissue fibers in a background of abundant serous fluid. Several areas of microcyst formation can be seen (Antoni B type of tissue). (100 x)

operative course was uneventful and on the eleventh day the patient was started on oral feedings. The patient developed a peculiar, painless "cushion-like" swelling around the stoma which has persisted to date. She was discharged on the 27th postoperative day with a No. 7 laryngectomy cannula in place. Immediate psychological adjustment was poor. Since discharge, the patient has been seen at frequent intervals and her mental outlook has improved considerably. She has gained a substantial amount of weight, continues to use a laryngectomy tube and is developing an esophageal voice.

Pathological report: (Fig. 1) The larynx was completely filled by a huge, ovoid partly encapsulated mass which measured 7 cm in diameter, which bulged prominently both posteriorly and laterally, extending also through the thyroid cartilage. The surface of the tumor was covered by smooth, pink, non-ulcerated mucosa. The consistency was firm and it cut with a moderate degree of resistance. Cut surfaces showed a tan, shaggy uniform structure firm in some areas but definitely softened and cystic in others. The attached adjacent tissue was searched for lymph nodes but none were found.

Several small nodular projections in this tissue appeared to be extensions from the tumor itself.

Microscopic diagnosis: (Fig. 2) Neurilemmoma. The tumor consisted of closely packed cells with spindle shaped nuclei (Schwann cells) and delicate reticulin fibers arranged in cords and less frequently in palisade formation. In other places the arrangement of Schwann cells was very loose, being separated from the fine connective tissue fibers by abundant amounts of serous fluid (Antoni B type of tissue). In the latter could be seen scattered cysts of varying sizes and occasionally areas of hemorrhage and necrosis. Blood vessels were few and confined to the periphery of the tumor.

SUMMARY

An unusual case of neurilemmoma of the larynx has been described. It represents not only the largest one ever described in this organ but also the only one treated by laryngectomy. The size and location of the tumor, as well as the precarious condition of the larynx and upper tracheal cartilages made this radical approach necessary. Thirty-nine other reports of laryngeal neurofibromas and neurilemmomas have been reviewed from the world medical literature.

REFERENCES

1. Baldenweck, L. and Leroux, Robert J.: Un cas de neurinome du larynx, Ann. d'Otolaryngologie 91 (Apr.-June) 1942.

2. Calvet, J. and Claux, G.: Le Neurinome du Larynx, Rev. de Laryngologie 69:47, 1948.

3. Claus, G.: Demonstration d'une preparation d'un neurinome du larynx, Soc. O. R. L. de Berlin 5; seance du 17-1, 1930.

4. Colledge, L.: Two Tumors of the Peripheral Nerves, J. Laryngol. & Otol. 45:409, 1930.

5. Eigler, G.: Drei seltene Geschwultse der oberen Luftwege, Hals-Nasen-u. Ohrenarzt. (Teil 1) 28:320, 1937.

6. Figi, F. A. and Stark, D. B.: Neurofibroma of the Larynx; Presentation of Five Cases, Laryngoscope 63:652, 1953.

7. Fisher, G. E. and Odess, J. S.: Solitary Neurifibroma of the Larynx (Report of Two Cases), Laryngoscope 59:1345, 1949.

8. Gignoux, A.: Presentation d'une malade atteinte de Schwannome du larynx, Soc. O. R. L. de Lyon (6 Juill.) 1936.

9. Helg, J. R.: Les Neurinomes du Larynx et la Neurofibromatose de Recklinghausen, Pract. Oto. Rhino. Laryngol. 12:165, 1950.

10. Holinger, P. H. and Cohen, L. L.: Neurofibromatosis (von Recklinghausen's Disease) With Involvement of the Larynx. Report of a Case, Laryngoscope 60:193, 1950.

11. Holmgren, G. and Bergstrand, H.: Case of Neurinoma Laryngis, Acta Oto-Laryngol. 12:514, 1928.

12. Hoover, W. B.: Benign Tumors of the Larynx; Their Diagnosis and Treatment, Surg. Clin. of N. A. 20:697, 1940.

13. Jackson, C. and Coates, G. M.: The Nose, Throat and Ear and Their Diseases, W. B. Saunders Co., Philadelphia and London, 1928.

14. Jackson, C. and Jackson, C. L.: Diseases and Injuries of the Larynx, The Macmillan Company, New York, 1942.

15. Lemaitre, F., and Ardoin, G.: Soc. d' O. N. O. de Paris (16 Janv.) 1936.

16. McHenry, L. C.: Neurilemmoma (Schwannoma) of the Larynx, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 62:392, 1953.

17. Malan, A.: Neurinoma della laringe, II Valsalva 13:417, 1937.

18. New, G. B. and Devine, K. D.: Neurogenic Tumors of Nose and Throat, Arch. Otolaryngol. 46:163, 1947.

19. New, G. B. and Erich, J. B.: Benign Tumors of Larynx; Study of 722 Cases, Arch. Otolaryngol. 28:841, 1938.

20. Oliver, K. S., Diab, A. E. and Abu-Jaudeh, C. N.: Solitary Neurofibroma of the Larynx, Arch. Otolaryngol. 47:177, 1948.

21. Pearlman, S. J., Friedman, E. A., and Appel, M.: Neurofibroma of the Larynx, Arch. Otolaryngol. 52:8, 1950.

22. Seidel, O.: Über eine sehr seltene Geschwulst des Kehlkopfes von unge- wöhulicher Grösse im Kindesalter (Neurinom), Arch. f. Ohren. Nasen. u. Kohl- kapfh. 148:210, 1940.

23. Slaughter, D. P., and de Peyster, F. A.: Pharyngeal Neurilemmoma of Cranial Nerve Origin, Arch. Surgery 59:386, 1949.

24. Smith, T. T.: Solitary Neurofibroma of the Larynx, Arch. Otolaryngol. 39:144, 1944.

25. Stout, A. P.: Tumors of the Peripheral Nervous System; Section II, Fas- cicle 6, Atlas of Tumor Pathology; Armed Forces Institute of Pathology, 1949.

26. Suchanek: Neurinomen des Kehlkopfeinganges Monatschr. f. Oh renheilk. u. Laryngo-Rhinol. 59:613, 1925.

27. Vail, H. H.: Schwannoma of the Larynx, ANNALS OF OTOLOGY, RHINOL- OGY AND LARYNGOLOGY 42:476, 1933.

28. Van Loon, E. L. and Diamond, S.: Neurofibroma of the Larynx, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 51:122, 1942.

29. Walker, J. S.: Neurinoma of the Larynx; Report of a Case, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 56:898, 1947.

(References 5 and 22 obtained in microfilm form through the courtesy of the Armed Forces Medical Library.)

XXXIV

LATERAL ABERRANT THYROID – A CASE REPORT

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The following case demonstrates an unusual manifestation of metastasis from carcinoma of the thyroid.

REPORT OF A CASE

A 36 year old white soldier was admitted to the U. S. Army Hospital, Nurmburg, Germany, with a six month history of an enlarging asymptomatic mass in the left side of the neck. He also complained of hoarseness and sore throat which had been present for three weeks.

Physical examination demonstrated a firm slightly tender encapsulated mass measuring approximately 7x5x5 centimeters situated medial and slightly anterior to the mid-portion of the sternocleidomastoid muscle. Several small nodes were felt in each anterior triangle. The vocal cords were inflamed and the laryngeal recesses contained mucopurulent secretions. Cordal motility was normal. The thyroid was not palpable. X-rays of the chest were normal. The solitary mass was believed to be a branchiogenic cyst. Surgery was postponed during the time that the suppurative laryngitis was treated. Excision of the mass was performed and examination revealed a thin walled, partially loculated cyst filled with a thin grey fluid. This fluid contained many cholesterol crystals but no tumor cells. The inner lining was smooth except in an area measuring approximately one square centimeter which was irregularly raised and papillary. The smooth area represented 95 per cent of the total wall and consisted of squamous and low columnar epithelial lining covering a thin fibrous layer in which were numerous lymphoid follicles. The raised regions contained papillary thyroid tissue. The pathologic diagnosis was "malignant metastatic thyroid carcinoma in a cystic structure."

A left radical neck dissection and a total excision of the left lobe of the thyroid was performed at the second operation. The thyroid

gland was of normal size and consistency. Sectioning demonstrated an area in the central portion of the gland measuring two millimeters in diameter which appeared grossly malignant.

Microscopically this proved to be an adenocarcinoma of the thyroid. No other foci were found in the neck bloc.

Malignant metastases in neck lymph nodes are generally found in solid masses. Solitary cystic tumors are usually benign. The case described indicates, however, that complete reliance cannot be placed upon gross morphology of a cervical cyst and that section for pathological examination is necessary for diagnosis. In this case sections from 95 per cent of the cyst wall were consistent with the clinical impression of bronchiogenic cyst. A study of the small papillary area led to the true diagnosis.

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A CASE OF BILATERAL EOSINOPHILIC GRANULOMA OF THE TEMPORAL BONE

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The literature is not voluminous on the subject of eosinophilic granuloma, and all published material was not readily available. Four papers, with their discussions were reviewed.[1-4]

Goodhill[4] in 1948 showed that Letterer-Siwe's disease, Hand-Schuller-Christian's disease and eosinophilic granuloma could be considered a histocytic granuloma of the skull, and a triphasic clinico-pathological syndrome. This statement is difficult to accept, however, when one reviews the characteristics peculiar to each condition.

Clinically, Hand-Schuller-Christian's disease is invariably associated with exophthalmos, polyuria, and mental deficiency. Abt-Letterer-Siwe's disease occurs before the age of two, and is invariably fatal. On the other hand, eosinophilic granuloma is a benign condition, and no less an authority than R. M. Hill,[5] feels that in eosinophilic granuloma of the bone spontaneous healing is the rule, with or without treatment. Jaffe and Lichenstein[6] have observed healing in these lesions which were verified by biopsy, and which were untreated. It must be concluded that Goodhill shows the presence of all three in one case.

Histopathologically, Letterer-Siwe's disease shows a marked proliferation of histiocytes in involved bone and many viscera, with varying degrees of superimposed acute inflammatory reaction. In Hand-Schuller-Christian's disease, the lesions have an accumulation of histiocytes which are usually filled with lipoid material. The lesions of eosinophilic granuloma are characterized by sheets and strands of histiocytes throughout which are conspicious collections of eosinophils.[6]

Radiographically, the lesions of eosinophilic granuloma reveal a localized, sharply-punched-out area of bone rarefaction, with no surrounding bone reaction.[7] Eosinophilic granuloma usually starts as a well localized lesion in the medullary cavity of a bone with a tendency to expand and perforate the cortex.[8] This probably ac-

counts for the exquisite pain at the site of the lesion, which is so frequently a symptom.

Circulatory eosinophilia of four to ten per cent is reported in some cases, and also an increase in blood cholesterol has been noted in others.

On exposure the lesion appears malignant, grayish-pink in color, soft and friable with a limited membrane and on cutting into the mass grossly suggests adenoma.

The following case is most interesting, in that only four cases of bilateral involvement of the temporal bone have been reported in the literature.[1, 2, 9, 10] H. E. Harris[11] of the Cleveland Clinic felt that the case was most unique because it occurred in an adult, since the majority of all reported cases occurred in the very young and in adolescence.

REPORT OF A CASE

M. D. D., a Master Sargeant in the U. S. Marine Corps, 34 years of age, was born in North Dakota on December 7, 1919. He was selected as a Naval Aviation Cadet at Chicago June 30, 1942, and in December, 1943, and again in April, 1950, qualified physically for the control of aircraft in group one. Each time he underwent a strenuous and complete physical examination, including blood tests, vestibular function tests, and audiometric examinations. His admissions to the sick list were for a varicocele, in 1944, at which time he was hospitalized for nine days; otitis media in 1946, hospitalized four days; surgical circumcision for congenital phimosis in 1951, not hospitalized; and urticaria, due to penicillin, in March, 1952, three days' hospitalization. He was given the usual innoculations, including cholera and yellow fever during his tour of duty.

On May 25th, 1953, as an out-patient of another naval hospital, a granulomatous mass was removed from the left external auditory canal, and the pathological report showed this to be a granuloma pyogenieum. On July 17, 1953, he was admitted to that naval hospital with the diagnosis of acute labyrinthitis, of the left ear. He was discharged four months and six days later with the same diagnosis. Complaints on admission were of headaches of four months' duration, and extreme vertigo, which he had had since the morning of admission. "He stumbled on walking and objects appeared to jump before his eyes." He had no nausea, tinnitus, or loss of hearing. Records of that hospital show that he had been under care for external otitis of the left ear as an out-patient for four months prior to admission, during which time he had had considerable antibiotic
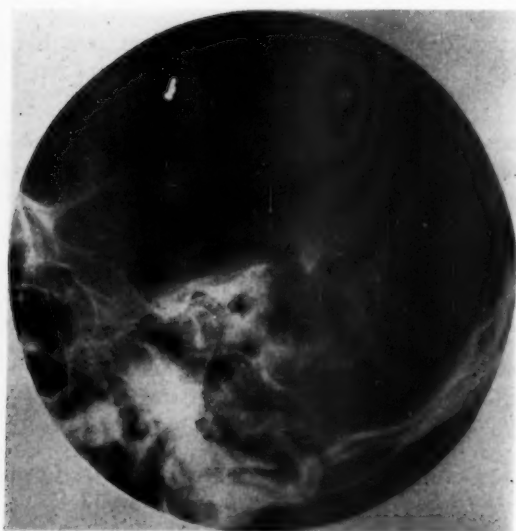
Fig. 1.—Disorganization of bone structure, right mastoid with bone destruction. (Jan. 7, 1954)

therapy. Examinations revealed a slight nystagmus. A small, granulomatous mass was found in the left external ear canal, on the posterior wall, and the external ear canal appeared edematous. A fistula test was positive in the left ear. There was a serpiginous rash on both buttocks. The granulomatous mass was again removed from the left ear canal on July 30, and reported this time as chronic otitis externa, etiology undetermined. The note from the pathologist records the gross appearance as a grayish-white tissue with a glistening gray membrane on one side. X-rays made July 17, 1953, showed cellular bone destruction of the left mastoid, with a normal right mastoid process. The interpretation was that of chronic mastoiditis confined to the tip cells on the left. The Kahn test was negative; the urine was normal. The white blood count was 9,200; hemoglobin 13.5 grams, or 93 per cent; hematocrit 48 per cent; sedimentation rate 32. The neurological consultant on July 21 reported no central nervous system disease, and considered that "the picture, for labyrinthitis, seemed bizarre." The ophthalmologist reported normal eye findings, including the fields. The dermatologist on July 27 reported the lesions of the buttocks as a fungus infection. An audiogram on

August 11, 1953, revealed an average 36 decibel loss for air conduc-
tion in the left ear, normal air conduction on the right, and normal
bone perception in both ears.   An exploratory left mastoidectomy
performed on August 26, 1953, "rapidly exposed the granulation
tissue and pus extending from the antrum into the tip cells and
through the inferior portion of the facial ridge into the external
canal.   The incus was inadvertantly removed."   A culture taken
from the surgical wound during the operation was reported as
staphylococcus aureus, coagulase positive.   A second culture taken
during the same operation was reported as "no growth."   Review
of the nurses' notes and doctor's orders revealed that the patient,
during this period of hospitalization, had had 600,000 units of peni-
cillin daily for 78 days.   He had had one gram of streptomycin daily
for 27 days and 300 mgm of dramamine daily for 25 days.   It is in-
teresting that there was no temperature elevation except on the first
postoperative day.   The blood count while in the hospital and prior
to surgery was normal, and a smear of the left ear canal on August
6, 1953, was reported as negative for bacteria.   The patient made
an uneventful recovery for a modified radical mastoidectomy and
was transfered to a second naval hospital for disposition on October
23, 1953.   Blood counts at this hospital revealed a 4 per cent and a
10 per cent blood eosinophilia.   Other examinations were within
normal limits.   He was discharged to limited duty on November 1,
1953.   Three days later, after a flight physical examination, the man
was placed in service group III, aviation.   It was noted that while
hearing was normal in the right ear, he had a loss of 50 debicels or
more at all frequencies in the left ear.

About the middle of December 1953, approximately one month
after his return to duty, he developed a vestibular disturbance of a
subjective nature, and had severe continuous headache in the right
temporal region.   He was admitted to the infirmary of the U. S.
Marine Auxiliary Air Station at Miami.   Two molars were found
to have "apical abscesses on the right side," and these were extracted
on December 31, 1953.   A popping sound had developed in his right
ear, and periods of total deafness ensued.   A physical examination
revealed external otitic infection in the right ear.   There was no
nystagmus found.   An audiogram on January 4, 1954, revealed
equal hearing loss in both ears.

On January 7, 1954, the patient was admitted to the naval hos-
pital at Jacksonville, Florida as a transfer from the infirmary in
Miami, with a diagnosis of deafness, right ear.   The above history
was confirmed, and the patient further stated that nausea and un-
steadiness has been severe for the first three days at the onset of his

Fig. 2.—Bone activity in operated left mastoid. Four months after surgery.

trouble one month previously. He stated that his hearing was satisfactory until ten days prior to his arrival at Jacksonville. The patient also stated that he had been receiving penicillin over a period of the past ten years. Each time he reported to sick call with complaints referable to his ears he had been given penicillin injections for a two or three day period; this had occurred at least once a year for the previous ten years. In 1952, prior to a rest leave in Japan, he was given an aureomycin capsule accidentally, and feels that that was the cause of the urticaria which admitted him to the sick list for three days at that time. In his mind this was verified by the fact that he had had two capsules of aureomycin in February 1953, which had caused swelling of his feet four hours after taking the second capsule. His protracted treatment for the ear condition, which led to the present admission, started in Miami in February 1953. He states that continuously at Miami, Chicago and New York while on duty he was given penicillin twice daily for periods of four to ten days at a time, at frequent intervals. In fact, he feels that he has had it almost continuously as an out-patient, for a year preceding the time of surgery. There is no record to confirm this, but the records since hospital admission on July 17, 1953 speak for themselves.

Physical examination on admission revealed an adult male, well-developed, well-nourished and well-oriented, with a complaint of difficulty in hearing and severe right temporal headache. The physical examination was negative except for his ears and the rash on his buttocks. The right ear was partially obstructed due to inflammation of the canal wall, considered to be an external otitis. The left ear revealed a retracted tympanic membrane, not inflamed. There was a left post-auricular mastoidectomy scar, and the left external meatus revealed an operative defect of a recent modified radical mastoidectomy. There was no nystagmus and no neurological abnormalities were noted.

Audiogram:	250	500	1000	2000	4000	8000
Right:	25	35	40	40	55	45
Left:	45	45	50	65	90	80

Bone conduction was excellent in both ears except for a bilateral dip to 40 decibels in both ears at the 4000 frequency. Routine laboratory examination was within normal limits, and x-rays were interpreted by the radiologist as showing a possible recurrent mastoiditis around the operative defect in the left ear (Fig. 1). The right mastoid process showed evidence of sclerotic mastoiditis with bone absorption and a break in the tegman tympani (Fig. 2). An x-ray ten days later showed a complete disorganization of bone structure with areas of bone destruction in the right mastoid.

It is interesting that by January 20, at the time of the second x-ray, the external otitis had resolved, and that the headache and dizziness had disappeared by February 10. Treatment here had consisted of propadrine(25 mgm four times a day, and wet Burrows wicks in the ear canals changed at frequent intervals. On February 17, 1954, the x-rays from the first naval hospitalization were received, and it was noted that the bone destruction in the right mastoid process had occurred since September 19, 1953, or in four months' time.

Caloric tests done on February 19, 1954, showed a normal response in the left ear and a dead labyrinth in the right. This was verified by repeated and stronger tests. Visual fields were found to be normal. On March 3 the patient was complaining of pain around his right ear and in the right cervical region. Examination of the right ear at this time revealed a protrusion of epithelium in the posterior portion of the canal 2 mm in diameter. This was thought to be a herniation from material of some sort coming from the facial ridge and recalled the previous findings in the left ear. Caloric tests were unchanged.

Fig. 3.—Circumscribed bony defect at inferior tip of scapula. (June 11, 1954)

Audiogram revealed an improvement in the air conduction for the lower tones in the right ear. X-rays at this time were interpreted as "progressing bone destruction." On March 4, a mastoidectomy was performed on the right ear, and an adenomatous mass was found occupying the substance of the mastoid process. This mass was under considerable pressure as evidenced by its herniation through the surgically uncapped mastoid cortex. The mass extended from the aditus ad antrum and the tegman tympani where there was found a dehiscence into the middle fossa one square cm in diameter, and it had eroded the facial ridge into the external ear canal, laterally to the facial nerve near the tip of the mastoid process. Posteriorly it had eroded the matrix to the surface of the lateral sinus. A dehiscence was not found in the bony labyrinth, and in order to preserve hearing, the contents of the epitypanum were not disturbed. Cultures taken from the mastoid cavity at the time of surgery were reported as showing no growth, and the second as staphylococcus albus. Microscopic studies were reported as showing eosinophilic granuloma. These tissue reports were confirmed by the National Institute of Pathology, Washington, D. C. The operative wound healed by first intention. Blood cholesterol studies done following the operation on March 12, showed 283.1 mgm per cent, and on March 23 a repeat study showed 304.2 mgm per cent. (The upper limit of normal blood cholesterol is 250 mgm.) Glucose was found to be 91.2 mgm per cent. Bence-Jones protein negative, N.P.N. 36. A bone survey by the radiologist on March 17, was not considered significant. X-ray therapy to the right and left mastoid was instituted on March 26, and intermediate voltage 140 K.V. with a total 1200 r to each side had been administered by May 13, a period of about six weeks.

The man, apparently in good condition, was returned to full duty on May 20, 1954 under medical observation. On June 11, the patient noted pain in his right shoulder, localized over the inferior tip of the scapula. A tender circumscribed swelling was present at this site and x-rays revealed a "lytic like tumor" at the inferior tip of the scapula (Fig. 3). X-ray therapy gave immediate relief and 900 r intermediate therapy was administered from June 12, through June 22. On July 2, the patient noted pain in the right mandible and x-rays again revealed bony destruction considered to be eosinophilic granuloma. The dentist reported the clinical picture to be similar to a "dry socket." (Fig. 4) Intermediate x-ray therapy immediately controlled the subjective complaints and a total of 900 r in air was administered from July 2 to July 16. The dentist reported the lesion to be granulating satisfactorily a few days after the first x-ray treatment. On July 15 the patient developed pain in the left pubic region. X-ray revealed bony destruction of the left pubis near the
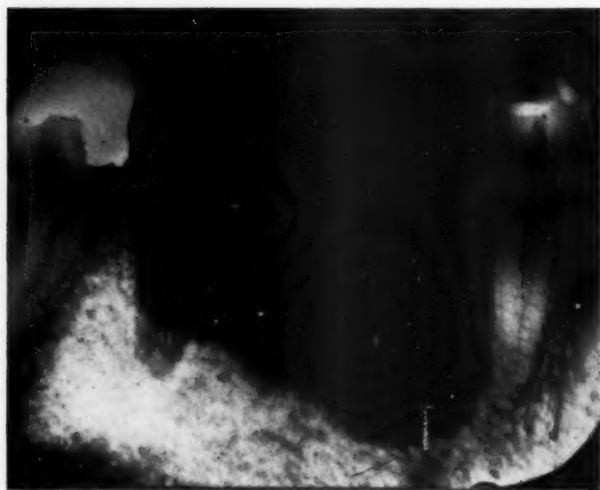
Fig. 4.—Bone absorption, right mandible, anterior to molar. (July 2,1954)

symphysis. Intermediate therapy to this area totaling 450 r from July 16 to July 21 promptly relieved the subjective symptoms. This therapy was not completed as the man was transferred to a facility where he could if required, have the advantage of systemic radiations with radioactive isotopes. In reviewing the first skeletal bone survey of March 17, 1954 there is evidence of the three lesions involving the right scapula, right mandible and symphysis pubis. However in review and in retrospect it is not difficult to see how they were missed. A re-survey of the skelatal system on July 20, 1954, showed "no new lesions; treated lesions have responded well with cessation of destructive processes and formation of new bone."

SUMMARY

1. A case of eosinophilic granuloma occuring bilaterally in the temporal bones of an adult, with a recorded medical history covering a period of twelve years, is presented.

2. The misuse of an antibiotic in this case is recorded.

3. It would appear reasonable to consider as a possible etiological factor a reaction to medication resembling epidermophytosis. This condition has existed on this man's buttocks for at least the past

year in spite of considerable dermatological care. Edema of the ear canals is recognized by physicians as frequently being caused by systemic use of antibiotics. Specific granulomatous lesions may result from allergic reactions.

4.   While these facts might not coincide with a true picture of allergy, the aggravation and extension of the eosinophilic granuloma to new sites, as well as renewed activity in the operated left mastoid following 47 million units of penicillin could suggest a reaction to the drug.

5.   The writer feels that there is evidence in this case, at least, that radiation therapy is the treatment of choice up to the present time.   It is further felt that radioactive isotopes may fill a need in the therapy of eosinophilic granuloma and its allied conditions known as the Letterer-Siwe's disease and Hand-Schuller-Christian's disease.

U. S. NAVAL HOSPITAL.

REFERENCES

1. Schuknecht, H. F. and Perlman, H. B.: Hand-Schuller-Christian Disease and Eosinophilis Granuloma of Skull, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 57:643-676 (Sept.) 1948.

2. Harris, Harold E. and Spencer, David M.: Bilateral Mastoid Involvement, Clev. Clin. Quart. 19:2 (Apr.) 1952.

3. Meredith, J. M. and Price, W. T., Jr.: Eosinophilic Granuloma of the Skull, Report of Four Cases of a Benign Lesion, Va. Med. Month., Vol. 79 (Jan.) 1952.

4. Goodhill: Victor: Histiocytic Granuloma of Skull, Laryngoscope 60:1 (Jan.) 1950.

5. Hill, R. M.: Non-Specific (Eosinophilic) Granuloma of the Bone, Brit. J. Surg. 37:69, 1949.

6. Jaffe, H. L. and Lichtenstein, L.: Eosinophilic Granuloma of the Bone, Arch. Path. 37:99, 1944.

7. Osborne, R. L., Freis, E. D. and Levin, A. G.: Eosinophilic Granuloma of the Bone: Presenting Neurological Signs and Symptoms; Report of a Case, Arch. Neurol. and Psychiatry 51:452, 1944.

8. Baker, G. S. and Fisher, R. G.: Eosinophilic Granuloma of the Skull; Report of Four Cases, with Five-Year Follow-Up, Surg. Clin. N. Amer. 28:1057, 1948.

9. Shea, J. J.: Xanthomatosis (Schuller-Christian Disease); Report of a Case with Radiosensitive Pathology in the Mastoid, Laryngoscope 48:589 (Aug.) 1938.

10. Wood, V. V.: Bilateral Xanthomatosis (Lipoidosis) of the Mastoid; Case Report, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 46:991 (Dec.) 1937.

11. Harris, Harold E.: Personal Communication. on March 24, 1954.

12. Hansel, French K.: Clinical Allergy, p. 117.

13. Hansel, French K.: Clinical Allergy, p. 346.

Notices

SIXTH INTERNATIONAL CONGRESS OF
OTOLARYNGOLOGY

General announcements and invitations to participate in the Sixth International Congress of Otolaryngology to be held in Washington, D. C., May 5-10, 1957, will be mailed to all otolaryngologists throughout the world within the month.

The program of scientific papers will be high-lighted by invited speakers on three principal subjects to be presented before the Plenary Sessions. The subjects will be announced shortly.

A motion picture program, scientific and commercial exhibits and various social functions are being arranged.

IV INTERNATIONAL CONGRESS

There are a few copies of the Proceedings of the 4th International Congress of Otolaryngology still available for purchase.

The publication contains all of the numerous papers, and is well produced. Copies may be had, at Five Guineas, from

> The Secretary
> British Medical Journal
> B. M. A. House
> Tavistock Square
> London, W. C. 1.

PORTUGESE OTORHINOLARYNGOLOGICAL SOCIETY

On March 20 the Sociedade Portuguesa de Otorrinolaringologia e de Bronco-esofogologia was founded, and held its first meeting in Lisbon under the presidency of Dr. Alberto Luís de Mendonça. The Secretary is Dr. António da Costa Quinta, Avenida de Liberdade 65, 1° Lisbon.

AMERICAN LARYNGOLOGICAL ASSOCIATION

CASSELBERRY PRIZE

A sufficient sum having accrued from the Casselberry Fund for encouraging advancement in the art and science of Laryngology and Rhinology, this sum is now available, in part or as a whole, for a prize award.   Theses must be in the hands of the Secretary, Dr. Harry P. Schenck, 326 South 19th Street, Philadelphia, Pa., before March 1, 1956.

Copies of the Transactions of the American Laryngological Association are available for general distribution at $8.00 a copy. Please send request with check to:

> Dr. Edwin N. Broyles,
> Editor Transactions
> 1100 North Charles St.
> Baltimore 1, Md.

UNIVERSITY OF PENNSYLVANIA

Announcement is made of the personal fundamental course in Bronchology, Esophagology, Gastroscopy and Laryngeal Surgery at the Graduate School of Medicine, University of Pennsylvania. The course begins September 8, and continues through September 19.

Courses are scheduled usually in January, June and September avoiding conflict with the meeting dates of the National Medical Societies.

Application may be made to Dr. Aims C. McGuinness, Dean, or to Dr. Gabriel Tucker, Chairman of the Department of Bronchology, Esophagology, and Laryngeal Surgery.

TULANE UNIVERSITY OF LOUISIANA
SCHOOL OF MEDICINE

The three year residency in otolaryngology offered at Charity Hospital of Louisiana at New Orleans on the Tulane University of Louisiana School of Medicine service is designed to qualify the holder for the examinations of the American Board of Otolaryngology and the practice of all phases of otolaryngology and endoscopy.

Candidates must be graduates of a class A medical school and must have completed a minimum of one year of general internship. An additional year of residency in internal medicine or general surgery is desirable but not essential.

All work is under the direct supervision of members of the Tulane Department of Otolaryngology, who are also members of the Charity Hospital Otolaryngological Staff; they are available at all times for instruction and guidance. Basic sciences are offered throughout each year of the residency during the academic year. The resident also participates in the program of the Speech and Hearing Center at the Tulane University School of Medicine.

The hospital year extends from July 1 of one year to June 30 of the following year.

Applications should be addressed to the Chairman of the Department of Otolaryngology, Tulane University of Louisiana School of Medicine, 1430 Tulane Ave., New Orleans 12, Louisiana.

HOME ACADEMY STUDY COURSES

The 1955-1956 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Detailed information and application forms can be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minnesota. Registrations should be completed before August 15.

UNIVERSITY OF ILLINOIS

The Department of Otolaryngology, University of Illinois College of Medicine, announces its Annual Assembly in Otolaryngology from September 19 through October 1, 1955. This Assembly will consist of two parts.

Part I—September 19 through September 24, 1955, will be devoted to surgical anatomy of the head and neck, fundamental principles of neck surgery and histopathology of the ear, nose and throat. This week will be under the personal direction of Maurice F. Snitman, M.D.

Part II—September 26 through October 1, 1955, will be devoted entirely to lectures and panel discussion of advancements in otolaryngology. The chairman of this section will be Emanuel M. Skolnik, M.D.

Registration is optional for one or both weeks.

* * *

The next Bronchoesophagology Course to be given by the University of Illinois College of Medicine is scheduled for the period October 24 to November 5, 1955. The course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

TEMPLE UNIVERSITY

Temple University offers a postgraduate course in bronchoesophagology October 17-28, 1955 and a postgraduate course in laryngology and laryngeal surgery September 19-30, 1955.

These courses are to be given in the Department of Laryngology and Broncho-esophagology, Temple University Hospital and School of Medicine, under the direction of Doctors Chevalier L. Jackson and Charles M. Norris. The tuition fee for each course is $250.00. For further information please apply to Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.

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President: Dr. Frederick T. Hill, Professional Bldg., Waterville, Maine.
Secretary: Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.
Next examination: Richmond, Va., March 6-10, 1955.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION

President: Dr. Clyde A. Heatly, 11 N. Goodman St., Rochester, N. Y.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Sq., Philadelphia 3, Pa.
Meeting: Mount Royal Hotel, Montreal, May, 1956.

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President: Dr. Bernard J. McMahon, St. Louis.
Secretary: Dr. Harry P. Schenck, 326 South 19th St., Philadelphia, Pa.
Meeting: Mount Royal Hotel, Montreal, May, 1956.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Dean M. Lierle, Iowa City.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Meeting: Mount Royal Hotel, Montreal, May, 1956.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

Chairman: Dr. Francis W. Davison, Danville, Pa.
Secretary: Dr. Hugh A. Kuhn, Hammond, Ind.

AMERICAN OTOLOGICAL SOCIETY

President: William J. McNally, M.D., 1509 Sherbrooke Street West, Montreal 25, Quebec, Canada.
Secretary-Treasurer: Lawrence R. Boies, M.D., 90 South Ninth Street, Minneapolis 2, Minnesota.
Meeting: Mount Royal Hotel, Montreal, Quebec, Canada, May 11-12, 1956.

The American Society of Ophthalmologic and Otolaryngologic Allergy

President: F. Lambert McGannon, Lakewood, O.
Secretary-Treasurer: Michael H. Barone, M.D., Buffalo, N. Y.
Meeting: Palmer House, Chicago, Ill., October, 1955.

VI International Congress of Otolaryngology

President: Arthur W. Proetz, M.D., St. Louis, U. S. A.
Secretary-General: Paul H. Holinger, M.D., 700 North Michigan Ave., Chicago 11, Ill., U. S. A.
Meeting: Washington, D. C., May 5-10, 1957.

International Bronchoesophagological Society

President: Dr. Andre Soulas, Paris, France.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U.S.A.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Justo M. Alonso, Montevideo.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U.S.A.

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Secretary-Treasurer: John F. Tolan, M.D., 3419 47th Ave., N.E., Seattle 5, Wash.

Canadian Otolaryngological Society

President: Dr. G. Edward Tremble, Montreal, Can.
Secretary: Dr. G. Arnold Henry, 170 St. George St., Toronto, Ontario, Can.
Meeting: Lake of Bays, Muscoka, Ont., June 16-18, 1955.